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The Edward Stirling Lectures.[1]

LECTURE I: FACT AND FICTION OF FACIAL PLASTIC SURGERY.

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FORTY years beyond the life span of a great name in your University is far too short a period for any stranger to come and recite facts which you all well know. I come only with humility at the honour you place before me and hope that my few words in these two lectures will not be out of harmony with the purpose of those persons responsible for choosing this method to perpetuate the name of one of the parents of your medical school. Nevertheless, I would like to add something I have learned about Sir Edward Stirling. He was ever ready to recognize and to accept change in a way many lesser men cannot do. He knew and worked on the principle that medicine and its traditions are no more static than the social environment in which they are cast.

Though the title of my subject may sound a little brash, it is more than timely that something be said under such a heading, and I take permission in the belief that if Stirling were here he would echo with the Queen herself, "Let us venture beyond the safeties of the past".

[1] Delivered in Adelaide on June 3 and 5, 1958.

While the emphasis of medicine has been on its art rather than its science, we as a protected professional group have remained splendid in our ivory tower, free to ply this art on a public long conditioned to accept all without question. They have been thankful for our help and ignorant of when our limitations become the will of God. Our shortcomings have not seen the light of day and the profession at large has not been subjected to probing and public criticism. For this ministration the more fortunate in life have paid more dearly and willingly that the less fortunate could receive it as charity.

Over this period the community has been well served by the rigid interpretation and observance of a long-tested ethical code. One of the principal tenets of this code has been to withhold from the public matters of professional knowledge—that little dangerous learning. Closely associated with this is another ethical principle—the complete bar to any personal advertising. It is on this background that our public relations have been built up over the years, and we within the profession are protected that one does not steal a march on his neighbour and colleague by the methods of business. No one could decry the desirability of these principles as such, but we must all be aware that the policy of complete abstinence from public information is having some undesirable effects both on public welfare and on the day-to-day life of the profession itself.

As citizens we have some obligation to the time in which we live. Unfortunately, perhaps, we have been drifting toward the Welfare State, in which increased personal and social welfare have become a right. As a corollary to this citizens demand these rights. While we

talk more and more of modern scientific medicine, this they expect as a taxpayer's right. It is understandable, therefore, with a public more and more educated and read in science, fed to them through potent avenues of information on a competitive market, that perhaps they have some excuse to criticize a silent and somewhat aloof profession.

If we continue to chase off an increased desire for information on medical matters there can be only one answer. While we hold to the restrictive basis of feeding out information by the well-known over-guarded statements—"A medical spokesman says, or denies, this and that"—which are protective, equivocal and devoid of any hallmark of authority, it is little wonder that the chief medium of public expression, the Press, becomes less and less interested in such statements, impatient with us and ready to underline an increasing disregard for an erstwhile revered profession. If, at the same time in the field of business competition, vendors of patent medicines and other so-called "health" features exploit and play upon an avid public with distorted glimpses of things hitherto regarded as unfit for them, sensationalism is the result—sensationalism of the worst type, which plays on every human emotion. This is not information. It is misinformation, and misinformed people become as unreasonable in their demands as they are unfair in their criticisms. To impart true information is not easy. It requires some organized, positive and persistent approach to the whole question of our public relations. And surely the first stage of this is to have a profession itself informed, willing and cooperative to that end and beyond the attitude of thinking only on a personal basis that someone or other is stealing a march. That is, there must be some real sense of bigness if any worthwhile policy of public information is to succeed.

Day by day there is evidence that Australia has entered a phase in these matters, which has been noticeable in Britain in recent years and from which, I would suggest, that America is already beginning to emerge. There, some regular order in factual, clear and palatable public information on medical matters replaces what in the past we have been too ready to regard as the crude and the unrestrained. Those of you who read well-known lay literature to keep up to date in modern methods would say that there does persist an element of alarmist journalese. On the other hand, if you read certain other magazines, despite horrible pictures taken on the roadside (which I would suggest do serve some good) you must be very impressed, as I have been, by such as the lucid explanation of the state of Eisenhower's coronary circulation or the dysfunction of his bowel. Some of you may ask, "Is this necessary?", but that is a question which serves no purpose in the democratic state if the public wants it. The point I am making is that if the public wants it and gets it, it should be based on realism, truth and some simple form of unemotional explanation. The American information on "Asian 'flu" made our efforts look puerile, as perhaps they were.

In the past year or two every one of us has been very critical and annoyed for a time at what we have read in the Press or heard on the radio. But how passive we, the same people, rapidly become about sensational information making more and more full copy and hard cash for Press, radio and television. Why should the picture of a tack make front page illustration? Only because it is a tack removed from the lung of a child sent overseas and financed in a blast of publicity. Or a radio picture, more like a London pea-soup fog, of some hospital operating room procedure, which could have been better made in one of our own bush hospitals. What do we hear of the future of people rushed overseas in similar circumstances to undergo operation for a condition which our own best informed surgeons regard and advise as inoperable? Blue babies we have had *ad nauseam*, and so it goes on. And by no means last on such a list comes plastic surgery, which brings me to my subject.

We live in the age of plastic, and it has long since past the novelty stage to have a much surprised patient exclaim, "I did not come here for an operation; I want plastic treatment", or to be asked by relatives after operation, "How much plastic did you have to use, doctor?" Only last week a woman came along asking if plastic tubes could be fitted to replace her "Fallopian tubes" which had been removed. Such are the oddities of ignorance. Nevertheless, plastic surgery and plastic surgeons mean many different things to people inside as well as outside the profession.

It is, indeed, one of the oldest ramifications of medical practice. As long as there has been destruction there have been attempts by man to reconstruct the human frame. The word "plastic" is from the Greek word meaning "to mould". By surgeons of repute it is synonymous with reparative or reconstructive surgery of parts lost or distorted by malformation, injury or disease. Nowadays, by the simple process of supply and demand and the difference of temperament, imagination and training required for individuals to pursue the attention to detail which makes for success or failure in this work, it is one of the accepted arbitrary subdivisions of surgical practice. It is becoming increasingly clear, as Professor Paterson Ross indicated in one of his lectures here last year on "The Future of Surgery", that as the physician and biochemist take over much of what in the past has been in the surgical field, the future of all branches of surgery must lie in the further application of constructive procedures. There are no longer any technical limits to destructive surgery; but as soon as we start to make things it matters not whether it is repairing a common bile duct, an interventicular septum, an opaque cornea or the internal ligaments of a damaged knee joint, it is then that surgery really becomes difficult. Here lies the challenge of the future. Here too will lie the application of many lessons which plastic surgeons have been studying and applying with effect in regions more accessible and where for obvious reasons the demand for repair in the main must lie.

However, it is no sinecure to practise in a field in which public demands and expectations are something quite unreal to the present state of scientific knowledge. One could illustrate this in many ways, but in these lectures I wish to discuss only three of the common lay demands on the plastic surgeon. In each my plea is for reason and perspective.

1. First comes the young lady front-seat passenger, who has been through the windscreen, with the request: "I have come to have these scars removed; they say you can take them right away, doctor."

2. Next, the woman whose face has been partly destroyed over the years by a little rodent ulcer and perhaps a lot of radiotherapy, who says: "I know you do marvellous things these days and can make me better looking than ever I was."

3. And last, the menopausal matron with the simple request: "I just want to look young again, and I leave the details to you, doctor—you know best."

These are tall orders, fanciful brain children of wishful thinking and public misinformation. The first two demands for removing scars and restoring lost parts I shall cover tonight. The call for youth and beauty I shall leave for my next lecture.

Scar Disfigurement.

Faces scarred by common injuries of modern life make up the great mass of cases, where the pressure of lay people is unceasing for early restoration by what weekly women's papers call "modern miracle surgery". Everyone can quote the case of a patient they have heard of in whom "you would never know there had been anything wrong with her". The ordinary social significance and abhorrence of a badly scarred face are well known; but one meets an extraordinary personal variation of attitude to facial scars among the relatives, often depending on where blame can be laid. At one extreme the parent driver is rather like the mother of a burnt child. Whether she did or did not, by her own carelessness, let the child get to the kettle, she feels there are many who openly or inwardly believe she is to blame, especially her husband. There is no end to her penance and indulgence. She will save her last penny and travel the world to have the scars eradicated. This is the sacrificial approach; she often asks can she give some of her own skin. By contrast, if the blame for an accident can be laid at the driver of a bus or the other car, no limit is set. In parental eyes a disfigured child can

never be adequately compensated, and the child is exposed to an atmosphere of social recrimination and bitterness. The effects of acquired face scars on prospective or established marriage relations when the contract was made on an undamaged article are also far reaching. They range from over-solicitude and chronic sympathy to disinterest or desertion. Mothers of eligible daughters are invariable in their observation of what a pretty girl she was.

With the injured themselves self-pity in some degree is the rule. In many this is covered by a mask of nonchalance, while at the other extreme is the woman who adopts a "wilted lily" pose, never to be given up. For the rest of her days she carries two articles in her handbag—the mirror she uses far more than in her hey-day and her favourite snapshot of youth.

To all these personal complications litigation arrangements and third-party insurance claims add their pressure for early removal of recent scars. Everyone is anxious for an end to the incessant paper war and wrangling which can go on over financial compensation for scar disfigurement. Long after broken skulls or legs are forgotten, disfigurement is emotionalized to full capital advantage. Everyone grasps every advantage of living in the mechanized age; no one will accept any of its risks or the price. How disfigurement is assessed in terms of money I have never yet been able to fathom. One is often surprised by apparently fantastic or incongruous figures awarded in the courts on this score. I sometimes wonder if these are not but a yardstick of the relative vigour of members of the legal profession. Once when I asked an ex-patient why she was seeking a luxury nasal operation, she told me she made so much out of her face scars she thought she should spend some of it on herself.

It is an unfortunate coincidence that panic about scar disfigurement reaches its height a few weeks after injury. By this time early thankfulness for life itself or for what might have been worse have evaporated. Gratitude for a social system of prompt public hospital attention, eagerly sought and graciously given at the time, may be soured. Other injuries out of sight are out of mind. Blood, bruising and bandages are no longer in evidence, and wounds declared healed are exposed for all and sundry to scrutinize. The fact, however, is otherwise. Healing is far from complete. Unfortunately, this is the period when scars are at their worst and healing in its full sense yet has far to go. It is in this time of panic that lawyers are consulted, and they, like all lay people at the time, are shocked at what they see; one and all promise the earth from justice and they advise, "You must see a plastic surgeon at once". What can we do? What do we do?

The prime consideration in most of these cases is entirely one of appearance, and the object of advice and treatment therefore must be directed to the best result on that criterion alone. With this in view, management of these scar-disfigured people must be adjusted to two sets of cold fact. One concerns time and the normal behaviour of scar tissue; the other concerns the practical limits and realities of surgical procedures.

Let us be categorical under these two headings.

Normal Scar Resolution.

1. First we explain that time is the great healer, that the normal behaviour of scar tissue is to become less vascular and to fade, and that it will become far less obvious than the recently healed wound. Much of what appears as lumpy and ugly is due to inflammatory reaction and will slowly resolve. Hard areas will soften, scars will contract in dimension. How many lawyer's letters has one written on this basis. Time will be kindly and we must be patient.

2. The residual effects of normal resolution cannot be properly assessed until a few months later. Only then can we give worthwhile indication of likely residual disfigurement and whether or not we can reduce this. A waiting period also enables us to ascertain the highly individual factor of how people scar so that we can face with confidence the paradoxical choice of more ambitious or radical procedures for a higher standard of result in those who scar well on the one hand, or on the other hand to do a minimum, if anything, and gladly accept compromise for individuals who obviously scar badly.

3. There are many natural factors, some known and some of them unknown, which influence the quality of scar production. We know that age, skin texture and the direction of a scar have a profound effect on its quality, no matter how it is produced or by whom. Scars across tension lines in no way compare with those in normal crease lines, as noted in comparing a vertical with a horizontal scar line on the forehead, for example; or again if we compare an oblique scar in the bulk of the cheek with a vertical scar across the line of the mandible. It is an unfortunate fact that the sex and age group in which best results are sought are often the most difficult and unpredictable. Middle-aged people with wrinkled and relaxed skin and reduced keloid tendencies make far and away the best scars. Tension plays some part in the production of keloid, but in young females especially the erratic basis of scar hypertrophy and keloid reaction is still in the main an unsolved mystery. It is easy to rush in and make bigger, brighter and better keloids.

4. "If something is to be done ultimately, why not now?", they ask. "Do you want the best result or some compromise?", that is the answer. Premature interference in the stages of induration and inflammatory resolution gives equivocal results and may add insult to injury. It often means unnecessary operations.

5. "How long must we wait?" "The longer the better, but about three to six months." When it is apparent that improvement can be made it is unreasonable and rarely practicable to wait indefintely, but if in doubt one might well postpone decisions concerning operation yet another three months.

6. Everyone asks, "What should I do about the scars meanwhile?". I do not believe that active treatment serves any good purpose during this phase. Most people want to massage the scars, but in view of the already good blood supply of the face I see no reason, nor have we seen any evidence, that massage can aid the kindly effects of time. If any question of radiation therapy for hypertrophy is suggested, it too calls for negative advice. If it is to be used as a method of treatment, it is best used in a concerted plan when finite treatment is ultimately elected. Why irradiate scars which we are going to excise six months later? It is then that we may consider X-ray therapy, if at all.

Such is the normal behaviour of linear scars. All this requires lucid and confident explanation to oil the troubled waters and deflate the prevalent ideas of urgency and alarm.

Surgical Achievements.

Confining our attention to the common scar disfigurement resulting from road accidents, such as in front-seat passengers injured by impact and by broken glass, the chief principles are as follows.

1. Normal contours are more important than scars. Bumps and irregularities make shadows and highlights which cannot be camouflaged. The common contour defects are the stepped or serrated scars often resulting from haphazard conditions of primary repair, avoidable and unavoidable, or from organized hæmatoma. Any linear scar will contract in its length and if it is in the segment of a small circle it will bunch up the tissue it surrounds to make the ugly little jagged lumpy scars we commonly see from the undercut wounds of small splintered glass fragments, especially if these have been allowed to heal spontaneously. These are corrected by turning them into linear scars, or if larger, breaking them into angled linear segments.

2. Wide scars can be reduced to acceptable linear scars. If they are in natural lines and not crossing concavities, such as the lateral nasal groove, for practical purposes they leave negligible disfigurement.

3. Scars in unnatural lines and directions or which tend to web concavities can often be much improved by rearrangements involving flap interdigitation generally on the "Z" principle. This gives relaxation in the scar line at the expense of tension in an opposite direction. Long scars in unnatural lines may also be improved in this way, but not the vertical scar up the forehead. Many of these are best left alone if otherwise acceptable.

4. Those obvious scars adherent to deep structures, especially the face muscles, can also be improved by flap rearrangements or by a layer repair, ensuring a fatty plane beneath the skin wound.

5. The correction of major skin loss is not a prime consideration in these cases, but in general, as far as possible, we avoid patchy-looking free skin grafts. We prefer to use well designed local flaps, even if this does mean creating more linear scar. If free grafts have to be used, as on the eyelids or the nose, we try to arrange their margins in natural lines and wherever possible to use skin allied in texture to that of the face, e.g. post-auricular or neck skin.

It is a significant fact that much of what we do at an elective secondary procedure in these cases is exactly what we would elect to do under favourable conditions at a primary repair. Most of the primary work is done in public hospitals.

In view of the increasing demands for this work, the large amount of money which is spent on secondary corrective procedures, litigation costs and figures of compensation awards, it is high time that teaching and hospital organizations paid more heed to this problem than they do in general. Few hospitals are geared to the demands for definitive primary repair under the conditions which are indicated. I believe the biggest contribution of the plastic surgery unit at the Royal Melbourne Hospital has been the teaching and training of housemen in this direction, but it is yet far from ideal.

Thus many come and few are chosen for early operation to reduce face scar disfigurement. Nevertheless, there are some indications for earlier surgery. Scar contraction causing dysfunction may indicate early correction, and scar ectropion involving eye exposure is the classical example of this. Other indications are for foreign bodies, glass or ingrained dirt. Foreign bodies in general promote deep scar and reaction, and we would wait indefinitely for softening and ideal local conditions. Tar ingraining, often not noted at the time of the injury, is a major problem. The almost insoluble problem of a severe diffuse tar ingraining warrants every order of precaution and care in its early recognition and management. Even when not removed at a primary operation, we have had some dramatic results by thorough scarification of pigmented areas a few weeks later.

"Sandpaper Surgery" for Scars.

What an indignity to surgery this newspaper nomenclature has become! The truth about abrasive treatment for scars is as follows. The superficial layers of the skin can be removed by various methods, one of which is surface abrasion; surface regeneration quickly occurs from the dermis, a regeneration which is slower the deeper the plane of removal. Any skin irregularity or disfigurement such as ingrained dirt or tar, therefore, can be removed along with the superficial layers by dermabrasion, and provided this does not involve the deep dermis or subcutaneous regions the residuum will be quick healing and free of scar. However, by the same token, if the lesion is deep situated, it remains uncured or else the abrasion is of an order to produce the scar of secondary healing with all its attendant risks and complications. Its application, therefore, is limited indeed and in no sense the equivalent of its fantastic publicity value.

There is no doubt that much can be done to reduce scar disfigurement, but note the word "reduce", for we can only replace a scar by a scar. If our endeavours are to be successful we must insure that misinformed patients really understand this. They must understand, too, that any new surgical scar is in some degree prone to the same early phases of induration and reddening and that the optimum result is still months away. It is surprising how frequently wedding days are planned on the basis of these operation dates and the immediate transfiguration which is expected to follow.

Any surgeon who undertakes to improve a scar must first have a clear idea of what the patient himself is expecting from treatment. If he cannot match up to this preconceived idea or modify it, no matter what the technical order of the job he does, it will be a failure if the patient is disappointed, and after your best proud efforts his first comment will be, "But there is still a scar, doctor!" Decisions to operate on scar disfigurement must be made, therefore, only when the surgeon is satisfied that the patient understands the limits of operative procedure, the possible complications, the degree of improvement which can reasonably be expected, and the time which must elapse for maximum improvement to take effect. Results can be assessed only in terms of satisfied patients, and these will be few if we fail to take at least some amateur psychological approach to this common problem.

Repair of Major Face Defects.[1]

Another of our unhappy tasks is the frequent necessity to disillusion patients presenting with some hideous defect, the result of disease, and/or its treatment when they have for long been subjected to the widespread misconception that "they do marvellous things with plastic surgery these days, and you won't know yourself". On the contrary, there are very strict limitations to the order of worthwhile surgical reconstruction of gross defects, and this in itself must be taken into consideration whenever mutilating operations on the face are anticipated. It is one of the very factors deciding operability. There is a school of thought which says that in efforts to cure cancer, for example, we should do anything regardless of cost, but there are humanitarian considerations which in our more enthusiastic age and stage we may tend to overlook. There are things in life which can be worse than death, and those with the agents of tissue destruction at their command—scalpel or machine—must ask themselves: What are the prospects of cure and at what price? Can the defect or destruction we intend to create be lived with in reasonable comfort and expectancy of life? These questions have a very particular application to the face. If an abdominal surgeon finds malignant disease in vital parts which cannot be done without, his decision is easy. On the other hand, with malignant disease of the limbs he can amputate parts which can be done without. We cannot amputate the head, but we can remove very large parts of the face; however, for most individuals the resulting defects and disabilities are impossible to live with in ordinary physical and mental comfort if they cannot be repaired. It is a most unhappy situation to be faced with these patients when they have been given no proper explanation of just what is involved, or what, if anything, can be achieved toward his restitution. "You go to the plastic surgeons and they will fix you up." And the misguided patient really believes it is as easy as that.

I am not raising here the important surgical question of "Should a post-operative defect be repaired, and if so, when?" I only wish to draw attention to the fact that there is a practical question—can the defect be repaired? Especially in the cancer age group we can effect repairs of a very high standard so far as skin cover, lining surfaces, soft tissue or bone bulk are concerned. Middle-aged and elderly patients scar well; they generally have redundant surface tissues and they have outgrown keloid tendencies. A slight hypertension is often helpful to the radical design of local flaps. Good standards can also be reached in the reconstruction of the fixed form of a structure such as the nose, provided there is some supporting base for it. On the contrary, when we come to replace mobile structures we are strictly limited. It is true that we sometimes appear to be able to make a mobile structure when muscle loss or denervation is incomplete. The filling in of a defect in a circular muscle mass, around the eye or the mouth for example, may also give some effective function. The destruction of war-time injuries, gross as they may have appeared, were seldom as complete as those of a really radical surgical excision. This limitation to the reconstruction of mobile structures has two particular applications.

The Upper Eyelid.

Over an artificial eye we can make a structure which behind the camouflage of heavy horn-rimmed spectacles may pass as an upper eyelid substitute. The same defect

[1] Portions of this section are quoted from an article by B. K. Rank and A. R. Wakefield (1958), "Surgery of Basal-Cell Carcinoma", *Brit. J. Surg.*, 45: 531.

over an exposed eye is a very different proposition, for total reconstruction of an upper eyelid, so that the functioning integrity of the eye is still maintained, is an undertaking which requires much careful planning. I have recently seen this effectively accomplished in a man who had to lose the whole upper eyelid, as well as sustaining a facial paralysis necessitated by a radical gland field dissection. The lower lid was transferred to the upper lid defect and a lower lid substitute subsequently reconstructed from forehead tissue. This was all possible because of thought and planning at the time of the upper lid excision. All this contrasts with the state of affairs we have had when parents present a child who has lost both eyelids after radiation for an angioma. They have come full of expectancy because they have been told, "We will refer you to the plastic surgeons to have new eyelids made". This, mind you, with a baby in arms and radiation damage of incalculable extent to the eye itself, to say nothing about inhibition of future growth of the whole region. It is no help to say, "Something must be done for this child"—the question is, what? Even in the absence of an eye we are less inclined to become involved in time-consuming surgical reconstruction for any extensive or composite defects of the eyelids, the eye socket and the periorbital regions than we were in war years. This is because of what can be accomplished by modern prosthetic orbital substitutes artistically designed and made. These prostheses are excellent, and they generally pass unnoticed by casual observers. Patients who receive them are astounded and are full of gratitude and praise for the work. For the same reason we often advise the fitting of similar prostheses for the eye and eyelids, rather than a difficult reconstruction of a contracted eye socket, especially in elderly people. The socket remnants are removed and obliterated prior to the making of the prosthesis.

Massive Amputations.

Massive amputations of the lower face may present another insoluble problem. Really gross amputations of the jaw, chin, cheek and lower lips can be performed for infiltrating malignant disease. Similar ultimate defects may follow slow destruction of these regions by radiotherapy, with the added disadvantage of an avascular, fibrosed and eczematous periphery. Having spent untold man hours and patience on the reconstruction of such defects, we hold the view that many of these defects should be regarded as irreparable. Now if patients knew this it might well have a totally different bearing on the original treatment or advice. We can migrate large soft-tissue masses to make something which would not raise comment in a crowded tram; we can restore effective bony continuity of the mandible, but we cannot make mobile structures with sensation. We provide at best only a dead-weight uncontrollable mass of tissue, which gets covered with spittle and bears excoriation badly. One is not suggesting that initial management in these cases is wrong; it may well be the right treatment. The point is that these patients, who have got quite enough to put up with, should not be subjected to moral let-down or breach of faith. There is no place for treatment at all costs; many patients, I am sure, would be better to finish life with their disease, for natural compensations in the symptomology of untreated malignant disease are full of surprises. Moreover, of these unfortunate people who after a long history of failed treatment are submitted to radical excision operations, how many are cured? In our experience, very, very few —and at what cost?

There is a third practical limit to worthwhile surgical reconstruction on facial regions. This concerns the ear, where the finesse of natural contour far outstrips the best we can achieve by multiple time-consuming reconstructive procedures. Any plastic surgery department could always fill its beds with patients for ear reconstruction; but except for partial deficiencies these are generally a complete waste of time and money. All plastic surgeons have spent untold hours in this pursuit, but none are really proud of their efforts. Here again the reason for our recent disinterest in total ear construction is admiration for the great improvements in prosthetic ears. This is due to applied artistry, the developments of soft plastic materials and methods used in their preparation and colouring. These have not yet been exploited as much as they could be. Furthermore, we can direct effective operative procedures to the better fitting and wearing of these aids, which makes up for some of the deficiencies of plastic surgery, especially in relation to cancer patients.

We are faced here with an odd paradox—the use and artistic application of plastic materials, a new scientific innovation, to supplement and aid plastic surgery, a very old branch of surgical art. It is little wonder that lay people are somewhat confused by the term plastic surgery. One must state here that our interest in plastic materials is essentially for external use; our attitude to buried foreign materials I shall indicate later.

In the past few years we have shown a number of patients with these aids at clinical meetings. Unfortunately, perhaps, many patients are prone to demonstrate these things themselves; a certain type is always keen to fool his fellows in the bar. From this again there has been a wave of unenlightened conclusions, and again there is wide divergence between what can be done in fitting a prosthetic substitute to a well prepared and healed area so that junction lines can be made at natural lines or points easy to camouflage, and, alternatively, in fitting a prosthesis over some hopeless hole, unhealed, discharging and surrounded by bone necrosis, the indications of which are surgical dressings for months to come. Again there are technical limits to the replacement of mobile structures, not in the case of the eye region, for these are the best of camouflage prostheses, but certainly in the case of the upper lip. After radical destruction of the upper lip and palate I have yet to see a prosthesis, no matter how carefully designed and prepared, which the patient gladly suffers for long. Quite apart from the difficulties of their retention, the cadaveric feel of them generally makes eating unbearable. With only one exception, among the few of these patients we have had to look after, all have come back to some arrangement which they work out as a compromise to their own satisfaction with dressing, cellophane tape and brown elastic adhesive. We can sometimes provide a light plastic shield to give some sort of contour to these dressings.

Finally, I would cite the case of a man who had untold energy, time and trouble expended on his behalf, to unrelenting pressure that "something must be done for this man", to the usual accompaniment of moral inflation by the well meaning but ill-informed. When ultimately fitted with what objectively was more than a reasonably good substitute for his lost part, this proved a subjective failure. He committed suicide. One can well regard this as an extreme example of a man badly let down and a good reminder of the plea I again make for a realistic approach to these problems.

MALARIA IN THE HIGHLANDS OF PAPUA AND NEW GUINEA.

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ESTIMATES of the altitude limit of malaria have varied from 1100 metres (3300 feet), quoted by De Rook (1935), to 1650 metres (5000 feet), reported by Spencer *et alii* (1956). Other workers, including Christian (1947 to 1949), Clements (1936), Egerton (1954), Giblin (1950), Black (1954) and Metselaar (1957*a*), have reported the presence of malaria up to about 1650 metres and its absence above 2000 metres (6000 feet). The conception of 1100 metres as the upper limit of malaria has long been accepted as a working hypothesis, and on this are based the regulations controlling the employment of highland labourers in coastal areas. The Native Labour Ordinance (1953) of the Territory of Papua and New Guinea selects an arbitrary limit of 3500 feet.

Metselaar (1957*a*) reports that in the Wissel Lakes and Baliem Valley areas of Netherlands New Guinea malaria transmission has yet to be proved, although the vectors are

present in the latter area. Christian (1947 to 1949) confirmed earlier findings by Army authorities (Anonymous, 1943) that malaria is present and may become epidemic at altitudes over 1500 metres (4500 feet). One such epidemic was reported by Spencer *et alii* (1956) in the Mt. Hagen area at about 1650 metres. Christian (1947 to 1949) commented in his earlier reports that with the march of civilization into the highlands there was every chance that the malaria problem could become more severe. With the recent extension of European development in the highlands and the extension of motor roads, airstrips and plantations, this problem becomes one which demands our urgent attention. This paper is an attempt to assess the limits and pattern of highland malaria, the recent extension of the disease and its potential routes of spread and the possibilities of controlling this spread.

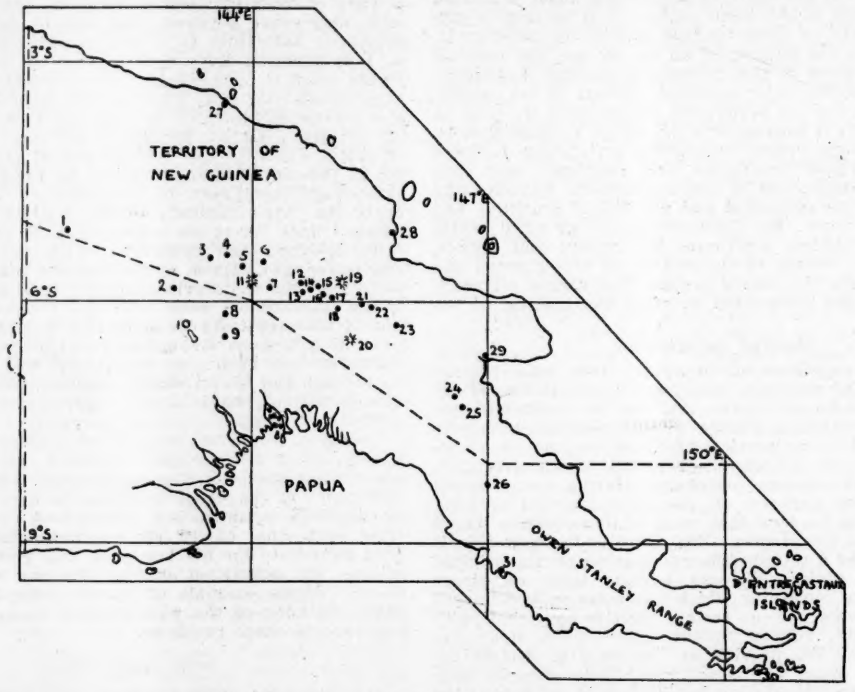

FIGURE I.

Sketch map of the Territory of Papua and New Guinea to show places mentioned in the text (excluding New Britain and adjacent island groups). 1. Telefolmin. 2. Tari. 3. Laiagam. 4. Wabag. 5. Wapenamanda. 6. Bayer River. 7. Mt. Hagen Station. 8. Mendi. 9. Erave. 10. Lake Kutubu. 11. Mt. Hagen, 13,120 feet. 12. Banz. 13. Minj. 14. Nondugl. 15. Kerowagi. 16. Mingende. 17. Chimbu. 18. Chuave. 19. Mt. Wilhelm, 15,400 feet. 20. Mt. Michael, 12,500 feet. 21. Goroka. 22. Benabena. 23. Kainantu. 24. Bulolo. 25. Wau. 26. Tapini (Goilala). 27. Wewak. 28. Madang. 29. Lae. 30. Samarai. 31. Port Moresby.

Geography and Climate.

The mainland of New Guinea and the main islands of the group are largely mountainous, except for a massive swamp area in the south. The highlands of the Territory of Papua and New Guinea are part of the New Guinea cordillera, which extends about 1500 miles from 3° S. in Northern Netherlands New Guinea to 10° S. in the d'Entrecastaux Islands. A branch of the cordillera runs north-east to form the mountain chains of New Britain and New Ireland. On the mainland, altitudes range up to 5130 metres (15,400 feet) at Mt. Wilhelm and even higher in Netherlands New Guinea. The cordillera varies in width from about 150 miles in the west to 25 miles in the Owen Stanley Range to the east (Figure I). Within the cordillera are numerous rivers and valleys, many of the latter being very extensive and almost completely isolated by high mountain ranges. Exploration of the highlands revealed large populations living in many of these valleys in a unique state of isolation. This geographical isolation has left many indigenes in a stage of cultural development akin to that of the stone age. Gilliard (1953) and Simpson (1955) have given good general accounts of the condition of these stone-age peoples.

The climatic conditions in the highlands vary considerably with local geographical features. The pattern and quantity of rainfall may show considerable differences with small changes in distance and altitude between one observation point and the next. From 80 to 200 inches (200 to 500 centimetres) of rain fall annually, mainly in the north-west monsoon. Mean temperatures generally fall with an increase in altitude and night frosts are common above 2300 metres. The approximate variation of temperature with altitude is as follows (Anonymous, 1951): at sea level the mean temperature is 81° F.; at 3000 feet, 69° F.; at 6000 feet, 61° F. (the theoretical minimal level for the development of the extrinsic cycle); at 9000 feet, 53° F.; at 12,000 feet, 44° F. At any given altitude the seasonal changes in temperature are slight, but the humidity at higher altitudes varies considerably from day to day, depending on local cloud movement and precipitation. The rainfall figures for Minj and Mt. Hagen at the east and west ends of the Wahgi Valley respectively are shown in Table I.

In both these localities there is perennial rainfall with the heaviest precipitation in the north-west monsoon. Data of this type give little information on the day-to-day rainfall pattern, on which depend the breeding potential and longevity of the local anophelines.

The Indigenes of the Highlands.

For the purpose of this paper indigenes of areas above 1300 metres will be referred to as highlanders. These fall into many ethnological groups and are concentrated mainly

TABLE I.
Rainfall at Minj and Mt. Hagen (Points).

Month.	Minj.						Mt. Hagen.				
	1951.	1952.	1953.	1954.	1955.	1956.	1951.	1952.	1953.	1954.	1955.
January	803	1492	740	1295	1280	1019	1126	1031	1249	1623	924
February	463	1144	880	1115	881	887	1079	1695	1205	883	862
March	806	899	951	1096	1002	1155	715	1628	1025	833	846
April	615	820	636	1164	1410	796	727	1284	897	1303	1785
May	372	296	528	934	1018	298	717	772	395	768	632
June	449	576	472	422	239	281	450	904	623	621	190
July	536	675	594	504	213	424	512	623	621	190	202
August	531	572	362	935	461	566	264	1104	445	835	908
September	1129	897	1081	850	739	618	1442	819	1119	725	733
October	512	906	471	583	776	580	540	790	701	801	760
November	1009	536	839	436	495	716	1094	1310	951	1013	729
December	662	908	853	1072	1137	1118	396	1175	424	1078	1327
Total	7887	9721	8407	10,370	9651	8458	9062	13,135	9575	10,267	10,154

in the Goilala, Goroka, Chimbu, Wahgi, Wabag and Tari Valley areas. Most highland houses are constructed in small isolated hamlets on the sides of the hills flanking these valleys rather than in the valleys themselves. The type of construction varies considerably from place to place, those in the Wahgi Valley being exceptionally small stick and grass huts. The domestic pig plays a large part in the cultural activities of most highlanders, and the periodical pig feasts are in many instances the only occasions for social intercourse of neighbouring groups (and no doubt also of their malaria parasites). Trade and the consequent movement of highlanders from one area to another are restricted by both lack of goods for trade and natural geographical boundaries, so that many tribes live in a strict seclusion with little connexion with even the adjacent valleys. Intertribal fighting has been an added factor in maintaining this isolation.

The highlanders are good primitive agriculturists and have extensive gardens both in the valley floors, which are in many instances extremely fertile, and on the hillside slopes. There is little wild game in the highlands and a resultant protein deficiency is seen. In spite of their near-vegetarian diet the general health and physique are good, but protein malnutrition takes a toll of young children and kwashiorkor is seen in some areas. Respiratory infections, skin diseases, dental caries, yaws and leprosy are the main endemic diseases apart from malaria. Intestinal parasites as far as is known do not play a major role in the disease pattern. All highlanders from areas above 3500 feet, who are recruited for work at lower altitudes, receive BCG, antipertussis and TAB vaccines before departure. On repatriation they are medically examined again, held in quarantine camps and given a course of antimalarial drugs (this will be discussed later).

The Anopheline Fauna of the Highlands.

The following species of anophelines occur in the highlands: *Anopheles annulipes* Walker 1856, *A. punctulatus punctulatus* Dönitz 1901, *A. punctulatus* intermediate forms (? = *koliensis* Owen 1945), *A. farauti* Laveran 1902, *A. bancroftii bancroftii* Giles 1902, *A. papuensis* Dobrotworsky 1957 (= *A. stigmaticus* of earlier authors). *A. punctulatus*, its intermediate form and *A. farauti* have been proved to transmit malaria in the highlands as elsewhere in the territory. The last two species have not yet been incriminated. One of the authors (S.H.C.) in 1956 recorded an over-all sporozoïte rate in *A. farauti* of 1·1% in 1019 gland dissections. He has also found *A. punctulatus* infected on a number of occasions. *A. farauti* occurs with some seasonal variation in density up to about 2000 metres west of Wabag, where Souness (1957) found it breeding in small ground pools. Lee (1946) reported that this species and *A. punctulatus* in the highlands attain a much larger size than their coastal counterparts. *A. farauti* is found in all the main valleys as far east as Kainantu. *A. punctulatus* and its intermediate forms are more seasonal in appearance, breed in temporary collections of water (in particular, man-made ground pools etc), and may spectacularly increase in density during intermissions in the rainy season. This species occurs up to between 1800 and 2000 metres, but for some unknown reason is apparently absent from the Goroka Valley, which is only about 1600 to 1700 metres. However, it does occur in the adjacent valley near Mt. Michael at 1800 metres. *A. papuensis* has been recorded by Lee (1946) up to 2300 metres near Mt. Hagen.

Many details of the bionomics of these vectors in the highlands are unknown so far. The natural longevity at different times of the year, the duration of the developmental stages, the anthropophilic index, the seasonal fluctuations in density and the sporozoïte rates and sensitivity to insecticides are at present being studied at the Malaria Section Research Unit at Minj.

Malaria Surveys in the Highlands.

The Altitude Limits of Malaria.

The most extensively surveyed area is the region between Kainantu in the east and Mt. Hagen in the western highlands. Further surveys have been conducted at Bayer River, Laiagam and Wabag in the north-west and at Tari and Erave in the southern highlands. Malaria is said to be present at Telefolmin in the Sepik highlands (Gunther, 1955). Clements (1936) recorded malaria at 1000 metres in the Goilala highlands and Egerton (1954) its absence at 2300 metres near by. The extensive surveys made between 1945 and 1949 by one of the authors (S.H.C.) showed that malaria was endemic in the Wahgi and Chimbu Valleys, but absent from the Goroka Valley. The Wahgi Valley findings have been confirmed by Giblin (1954), Black (1954) and Spencer *et alii* (1956). Malaria was found to be absent from Wabag and Laiagam, but present at lower altitudes in the Bayer River, Tari, Erave and Lake Kapiago (1400 metres). Speer (1957) found malaria there and Souness (1957) reported malaria north-west of Wapenamanda at 1900 metres. The results of the main surveys referred to are presented in Table II.

All three members of the *A. punctulatus* group have been recorded at Mt. Hagen (1700 metres) and nearby at 1900 metres by Lee (1946). From this data it appears that the upper limit of malaria lies between 1800 and 2000 metres (5500 and 6000 feet), depending on the exact locality. In all areas the endemicity is greater on the valley floors than on the hill slopes. The local variations are well shown by an analysis of some of the data summarized in Table II. The 1946 to 1947 Chimbu and Minj surveys show that there is a variation in incidence from Chuave in the east (spleen rate for adults is 0·8%; for children, 1·2%) to Minj in the west (spleen rate for adults is 10% to 17·8%; for children, 36·4% to 42·3%). These figures are summarized in Table III.

In an interpretation of these figures allowance must be made for the fact that these surveys were made consecu-

TABLE II.

Malaria Surveys in Highland Areas.

Date.	District.	Altitude. (Feet.)	Children.		Adults.		Author.
			Number Seen.	Spleen Rate. (Percentage.)	Number Seen.	Parasite Rate. (Percentage.)	
1945	Mt. Hagen ..	5000+	295	11·5	?	6·1	Christian.
1946 to 1947	Chimbu and Minj	5000+	3124	19·6	2954	5·7	Christian.
1948	Wapenamanda	5500	—	—	—	—	Christian.
1949	Goroka	5000+	8083	1·0	?	0·1	Christian.
1950	Baiyer River ..	3900	—	—	—	—	Christian.
1954	Tapini	3000	500	17·2	—	—	Egerton (1954).
1936	Goilala	5000 to 7000	618	0·0	—	—	Clements (1936).
1956	Tari	5250	76	10·9	76	1·3	Peters.
1956	Wabag	6500	69	0·0	69	0·0	Peters.
1956	Lalagam ..	6950	115	0·0	115	0·0	Peters.
1956	Erave	3700 to 5000	659 (all ages)	43·7	676	1·9	Jameson.

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1956	Wabag	6500	69	0·0	69	0·0	Peters.
1956	Lalagam ..	6950	115	0·0	115	0·0	Peters.
1956	Erave	3700 to 5000	659 (all ages)	43·7	676	1·9	Jameson.
					222	0·5	Christian.
					16,397	1·0	Christian.
					182	51·6	Christian.
					—	—	Egerton (1954).
					—	—	Clements (1936).
					156	0·6	Peters.
					216	1·3	Peters.
					106	2·0	Peters.
					—	—	Jameson.

tively in the different areas and not simultaneously. As will be shown later there is a considerable seasonal as well as annual change in the endemic indices. These local variations are due to the nature of the terrain, well-drained sloping areas providing less potential breeding sites than ill-drained valley floors with a high water table.

The difference in the intensity of malaria due to the exact locality of the habitations was illustrated by Black (1954). In May, 1954, he found the indices in Upper and Lower Minj Valley people shown in Table IV.

Spencer and Spencer (1955) made the interesting observation in the Minj area that women showed higher indices than men, and attributed this to the custom of men leaving the women to tend and live on the gardens of the valley floor with the pigs, while the men stay at home on the nearby slopes. However, one of the authors (W.P.) has also found this sex difference in a subcoastal holoendemic area where the men and women stay together.

Seasonal and Yearly Variations.

Malaria morbidity is distinctly seasonal in the Wahgi Valley. Analysis of the proportion of malaria cases to all admissions to hospital shows a seasonal increase between May and October, that is in the "dry" season. The figures for 1954 to 1956 also show that the peak differs from year to year and that in each case both *Plasmodium falciparum* and *P. vivax* are responsible, the latter dominating the picture in blood films. From the clinical standpoint, Spencer and Spencer (1957) found that *P. falciparum* malaria was dominant in epidemic periods. These data are illustrated in Figures II and III.

Unfortunately, we have very little data to illustrate the relationship of vector density to malaria prevalence. At Minj it has been observed that an increase of malaria cases follows the pullulation of *A. punctulatus* after intermissions in the rainy season. The epidemic of 1955 in the Mt. Hagen area reported by Spencer *et alii* (1956) followed about two months after a particularly heavy month's rainfall in April, 1955, which was itself followed by a relatively dry spell. However, during this particular epidemic only *A. farauti* was found. In Minj at the other end of the Wahgi Valley, 1954 was an epidemic year, with up to 55% of all patients admitted to hospital showing parasitæmia. Hospital records before this period were unreliable, but seem to indicate that 1952 had an unusually high malaria incidence. Black's findings at Minj in May, 1954, are summarized in Table IV. Metselaar (1957*b*) conducted a small survey at Minj two months prior to Black and found spleen rates intermediate between those of Black and those of Christian shown in Table III, which were recorded during a period of low endemicity. Spencer and Spencer (1957) found that at Minj in 1954 spleen rates in children aged two to 10 years varied between 23% and 91%. Adult spleen rates were up to 97% in one group. They examined a total of 8799 people living in 18 different social groups in various parts of the Minj Valley.

The Mt. Hagen epidemic was an interesting illustration of the ease with which the diagnosis of malaria can be

TABLE IV.

Survey Data, Minj Valley, May, 1954.
(Black, 1954.)

Minj Valley.	Children.			Adults.		
	Number Seen.	Spleen Rate. (Percentage.)	Parasite Rate. (Percentage.)	Number Seen.	Spleen Rate. (Percentage.)	Parasite Rate. (Percentage.)
Upper ..	69	6	6	359	9·2	3·6
Lower ..	28	65	23	106	75·5	25·5

TABLE III.

Surveys in the Chimbu-Minj Area by Christian, 1946 to 1947.

Area.	Approximate Altitude. (Feet.)	Children.		Adults.			
		Number Seen.	Spleen Rate. (Percentage.)	Number Seen.	Parasite Rate. (Percentage.)	Number Seen.	Spleen Rate. (Percentage.)	Number Seen.	Parasite Rate. (Percentage.)
Gogmei	5000	306	0·3	306	0·0	1068	0·8	477	0·4
Chuave	4900+	313	1·2	313	0·3	1132	0·8	565	0·0
Kundiawa	5100	510	11·3	510	0·9	1945	4·6	730	1·2
Mingende	5200	384	15·0	380	5·2	1217	6·8	500	3·6
Kup	5200	635	16·3	460	3·0	1634	5·3	925	1·8
Minj	5000+	423	36·4	423	17·0	2330	10·0	750	6·9
Bangi-Kerowagi	5210	553	42·3	553	10·3	2616	17·8	917	5·2

missed in an area of rather low endemicity when the disease flares up into a sudden epidemic. The actual epidemic probably built up over the previous month, during which time an epidemic of "influenza with pneumonic complications" was reported. Further investigation into the cases and a field survey in July, 1955, revealed up to 78% of "positive" blood films in an area normally mesoendemic. *P. vivax* was the parasite mainly responsible for this epidemic.

It is clear that in those areas of the highlands such as the Wahgi Valley where malaria is present it is of the "unstable" type, as defined by Macdonald (1953). This implies the following conditions according to this authority: (i) transmission by a vector which bites man relatively infrequently; (ii) a relatively high vector density (one to 10 or more bites per person per night); (iii) the possibility of "anophelism without malaria"; (iv) marked seasonal changes in response to changes in temperature and humidity; (v) marked fluctuations in incidence from gross epidemics to near elimination of the disease; (vi) varying immunity in the population; (vii) relative ease of control.

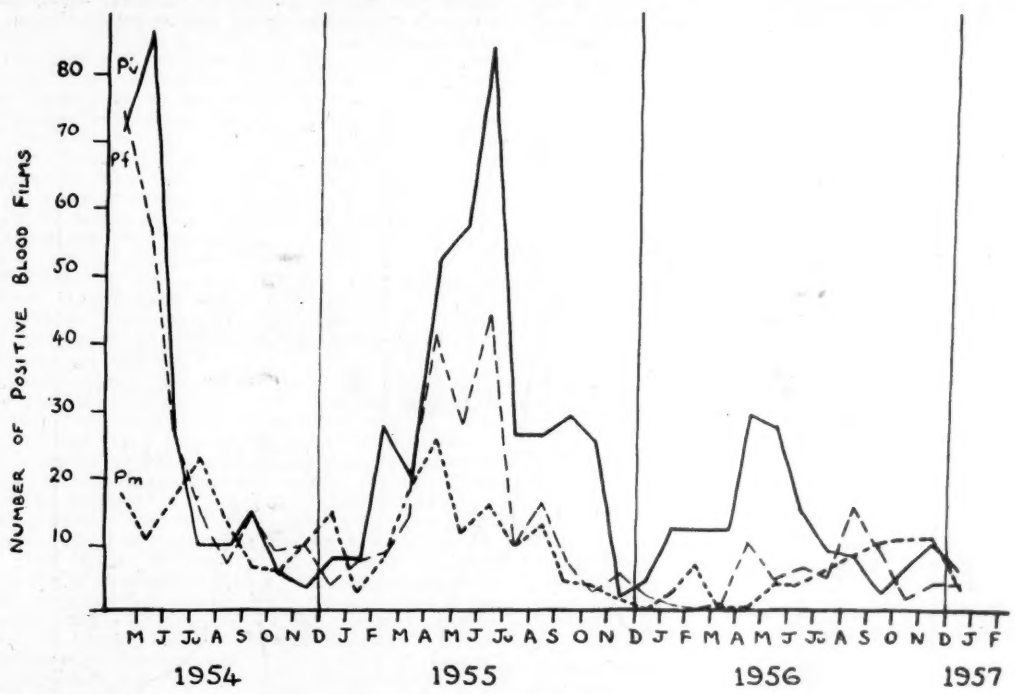

FIGURE II.
Monthly parasite incidence (Minj Native Hospital).

The Extension of Malaria in the Highlands.

There is some evidence that with an increase of European influence in the highlands malaria is extending into hitherto non-endemic areas. Recent surveys by one of the authors (J.L.J.) have revealed that malaria is now endemic in two areas of the Goroka Valley, shown in 1949 to be malaria-free, as stated earlier. At Kamaliki he found an over-all spleen rate of 5% in 110 people of all ages. Five of those with splenomegaly denied ever having left the valley and the sixth was a repatriated labourer. At Asaro a similar situation was found. At Lufa in the next valley to the south a mesoendemic situation was found, but there malaria appeared to have been long established.

The survey conducted by the same worker at Erave, a recently opened station in the southern highlands, produced data suggestive of a spread of malaria round the station, diminishing in proportion to the distance from the station. However, there was also a gradual increase in altitude which may have accounted for the change, and figures are too small to allow conclusions to be drawn. The data are summarized in Table V.

TABLE V.
Malaria Survey (All Ages) at Erave.

Village.	Distance from Erave.	Altitude. (Metres.)	Spleens.		Bloods.	
			Number Seen.	Spleen Rate. (Percentage.)	Number Seen.	Parasite Rate. (Percentage.)
Erave:						
Strangers ..	——	1230	114	14	116	0
Locals	——	1230	224	61	231	3
Iamurupi	Two miles.	1270	142	65	146	3
Kolari	Four miles.	1270	60	47	63	0
Batri	Half a day by foot.	1430	68	15	69	1
Kagua	Two days by foot.	1666	51	10	51	2

Erave is in steep, rugged, limestone country with good natural drainage in the hills, and village communities are considerably isolated by the nature of the terrain. The valley floors are swampy and the villages are sited within flight range of the vectors. In the course of building an airstrip new breeding sites admirably suited to the development of the *A. punctulatus* group were produced. Imported personnel were placed on chloroquine suppression two and a half months prior to this survey owing to their high incidence of sickness due to malaria.

In 1949 Christian (1947 to 1949) gave a warning that, with the advance of European settlement, bringing in its train both increased construction and movement in and out of the highlands of natives, there was every possibility that malaria would increase. At that time he could find no evidence that malaria had become established along the old trade route from the Markham Valley into Kainantu, although a few cases were imported. Neither could he find any evidence of spread from the endemic Chimbu Valley area to the nearby Goroka Valley. There was no suggestion that malaria had followed the stationing of troops at Bena during the war years in spite of the obvious danger. He believed that malaria had been endemic almost from time immemorial in the Wahgi Valley and Chimbu areas, probably reaching there via the Bomai salt flats to the south. The survey at Tari, which revealed a small number of indigenous cases of malaria, was also of interest since this area, which was opened about five years previously, now receives a number of outsiders from the hyperendemic Lake Kutubu area to the south. An examination of 17 Lake Kutubu porters at Tari, 1750 metres high, showed 14 with splenomegaly and three with parasitæmia (including one with gametocytes of *P. malariæ*). A recent survey in the Wabag-Wapenamanda area by Souness (1957) showed 2% of enlarged spleens in children up to nine years of age there and a few enlarged spleens further west at about 1900 metres. A comparison with Christian's 1948 survey suggests that malaria is now extending westwards towards Wabag.

The importance of minimizing the risk of spreading malaria by bad constructional and agricultural methods which increase the number of potential vector breeding sites was emphasized at the time of the Mt. Hagen report (Editorial, 1956).

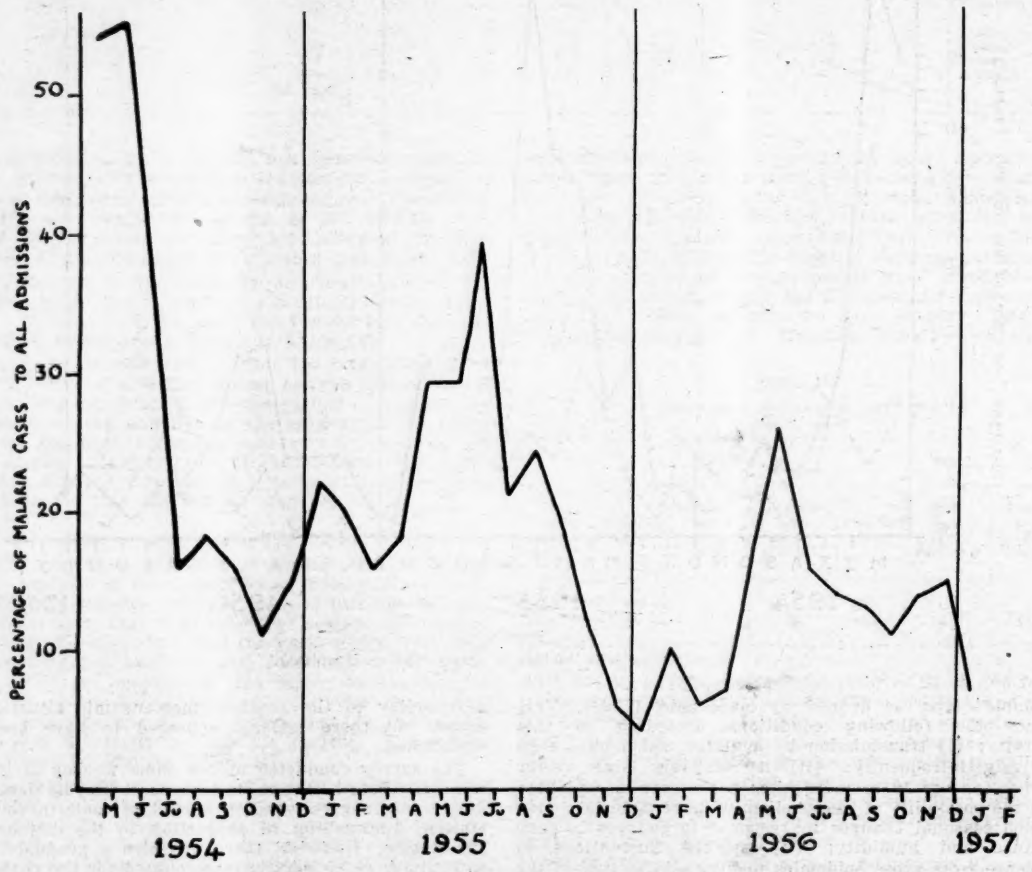

FIGURE III.
Monthly malaria case incidence (Minj Native Hospital).

Potential Precipitating Factors which Lead to an Extension of Malaria.

Macdonald (1953) has summarized the changes which may lead to an increase of malaria as follows.

1. The introduction of the malaria parasites.
2. The introduction of anophelines.
3. The numerical increase in anophelines after human activity.
4. The migration of non-immune people.
5. The deviation of anophelines from animals to man.
6. Climatic changes.

With the exception of item 5, all these points are relevant to a consideration of malaria in the New Guinea highlands.

1. There are a number of possible routes by which parasites may be introduced into new areas. Those are in order of importance: (*a*) labourers recruited for work on the coast; (*b*) government and other employees (e.g. mission and plantation personnel, domestic servants) entering the highlands from endemic areas; (*c*) troops entering the highlands (both European and native). The question of highlanders being transferred to endemic areas on the coast has already been mentioned, and their movements and medical care are prescribed by law. The details of their medical care will be mentioned later. There is no legislation controlling the entry into the highlands of partially immune people normally resident in endemic areas, and the accumulation of such people in European settlements, mission stations etc. may introduce a dangerous source of gametocyte carriers. The same may be said of troops entering the highlands, although we have already commented on the failure of malaria to spread from this source at Bena. An anonymous Army publication (1943) pointed out that infected troops, including Europeans, frequently suffered from relapses of malaria during the war when they moved into the colder mountain areas. The Japanese forces in particular, lacking adequate antimalarial drugs, suffered severely in this way during their attack on Port Moresby across the Owen Stanley Ranges.

2. There is no suggestion that any new species of anopheline vectors have been introduced into the highlands, although Peters and Standfast (1957) have shown that *A. karwari* is spreading into the Sepik district from Netherlands New Guinea.

3. As mentioned earlier and emphasized by a number of writers, the development of a new area by Europeans almost invariably leads to the extension of breeding sites, particularly for the *A. punctulatus* group. Road construction, airstrip construction and bad agricultural methods are all a potential danger and undoubtedly played a large part in the increase of transmission which appears to have taken place in certain areas of the highlands. In the Wahgi Valley itself, breeding was increased along the valley road from Nondugl to Mt. Hagen by the deterioration of roadside ditches, drains and borrow pits. In Tari new breeding places were formed during the construction of the airstrip and the same appears to have happened at Erave. Poor agricultural methods resulted in an increase in malaria at Korn Farm near Mt. Hagen. In 1945 Christian recorded there an adult parasite rate of 13% and a spleen rate of 21%. *A. farauti* was found at Korn, and at Mt. Hagen *A. punctulatus* and *A. koliensis* were also present. Labourers from other parts of the area were congregated at the farm for periods of about six months, during which time the increased anopheline population due to a bad drainage system must have extended the infection to many new hosts. These later dispersed to their homes, taking their infections with them. This may have been an important precipitating cause of the explosive epidemic around Mt. Hagen in 1955.

4. Non-immune natives migrating may suffer from malaria while in an endemic area and transmit it to their neighbours when they return to their homes. The latter is more likely after relapses of *P. vivax* and *P. malariæ* than in *P. falciparum* infections. It has already been mentioned that highlanders working on the coast receive regular chemoprophylaxis. By law they must be provided with one of the following drugs, and it is the responsibility of the employer to ensure that the drug is actually consumed: "Paludrine", 100 milligrammes a day; mepacrine, 100 milligrammes a day; quinine sulphate, seven and a half grains a day; chloroquine base, 600 milligrammes a week (one tablets four times a week); "Camoquin", 800 milligrammes a week (one tablet four times a week).

That this prophylaxis and the other measures taken to protect the health of highlanders are effective may be judged from the following unpublished figures. Between January 1, 1950, and October 31, 1951, 10,250 labourers from the central highlands were employed at lower altitudes, mostly on the coast. Of these, 4·97 per thousand died during their service. Of 30,826 natives from areas below 3500 feet employed on the coast during the same period, 5·22 per thousand died during their service. Without adequate protection against malaria there is no doubt that many of these highlanders would have succumbed to the disease in the highly endemic coastal areas where they were employed.

On repatriation to the highlands all labourers are held at quarantine camps for a period of two weeks, during which time they are medically checked and given a course of a malaria schizonticide *plus* one of the eight aminoquinolines. That there have been some failures in prophylaxis during their employment is revealed by the following figures obtained from the Goroka quarantine station. In July to September, 1956, 1201 repatriates examined showed 4·7% hepatomegaly and 19% splenomegaly. In December, 1955, to September, 1956, of 2831 repatriates' blood films examined, 0·3% yielded positive results (*P. falciparum*, nine; *P. vivax*, one). During quarantine repatriates developed infections as follows: *P. falciparum*, 65; *P. vivax*, eight; *P. malariæ*, one; mixed falciparum and vivax, and mixed vivax and malariæ infections, one of each. These repatriates each received a fourteen-day course of "Quiniplex", totalling 900 milligrammes of quinine sulphate and 39 milligrammes of pentaquine phosphate a day. The relapse rate of the New Guinea Chesson strain of *P. vivax* is high on this regime, and some of these highlanders may thus have taken their parasites back to their homes.

This course has been replaced by a combination of 2·1 grammes of "Camoquin" and 0·28 gramme of primaquine given over a two-week period (that is 150 milligrammes of "Camoquin" and 20 milligrammes of primaquine a day). This will be administered at the existing quarantine camps at Goroka and Tapini and at the new camp to be established at Mt. Hagen. The dosage of primaquine is higher than that generally prescribed in other countries, owing to a clinical impression that the standard dose of 15 milligrammes is insufficient against the New Guinea strains of *P. vivax*. Careful observations have shown no evidence of toxicity at this dosage. The dose of "Camoquin" will be reduced in the future as it is no longer believed that such a prolonged administration of this drug is necessary.

5. The deviation of anophelines from animals to man is unlikely to play any part in the extension of malaria in the highlands, where the only domestic animals of any importance are the dog and the pig.

6. Climatic changes play a considerable part in the causation of epidemics and have already been discussed.

Discussion.

It is clear that the old concept of 1100 metres as the altitude limit of malaria is no longer tenable, but the proposition that the limit for legislative purposes should be raised is debatable. Malaria occurs above this altitude up to nearly 2000 metres, but, as pointed out by Spencer and Spencer (1955), the frequency of transmission is too low to enable the population to develop a high degree of immunity.

There is some evidence that malaria is extending into new areas, along with the development by Europeans of those areas. This has been precipitated partly by the manufacture of new breeding grounds especially for the *A. punctulatus* group. This species may be responsible for seasonal epidemics, while *A. farauti* maintains a perennial low level of endemicity. Malaria in the highlands is thus very unstable (as compared with the stable malaria prevalent in the lowlands), and dangerous epidemics are a constant threat in borderline areas. Geographical and seasonal variations make the task of comparing data from place to place and time to time very difficult, and more careful and regular observations are required on the entomological and clinical aspects, especially infant parasite rates, to clarify the picture of malaria endemicity in the highlands. Such work is at present being conducted at the Malaria Section Research Unit at Minj.

There are undoubtedly many potential paths of spread, and that this spread has so far been very limited requires explanation. The following are among the possible limiting factors: (i) the cool climate, which may limit the development of the parasites in the vectors; (ii) the dispersion of the population in small hamlets rather than their conglomeration in large village groups; (iii) a possible altitude factor inhibiting the development of the pre-erythrocyte cycle as postulated by Freyvogel (1956); (iv) tribal isolation due to intertribal fighting, which still continues in some outlying areas.

Control Measures.

The control measures that should be or are being taken to minimize the extension of malaria into the highlands are as follows.

1. The control of man. (i) Chemoprophylaxis in all highlanders employed in coastal or low-lying areas should be enforced; chemoprophylaxis in those employed in the highlands is also desirable, especially during seasonal epidemics. (ii) A more reliable course of antimalarial therapy for repatriates is needed. (iii) All coastal people entering the highlands should be treated with primaquine, especially infants and young children. (iv) Primaquine should be in general use in hospitals for the treatment of gametocyte carriers.

2. The control of vectors. (i) All activities such as road and airstrip construction, land reclamation and agricultural development should be controlled to minimize the production of anopheline-breeding sites. (ii) The instruction of highlanders in improved agricultural methods is needed. (iii) Well-maintained and supervised drainage schemes, where applicable, on the pattern of the now abandoned pilot drainage scheme at Banz are necessary. (iv) The general education of all administration personnel, European settlers and the indigenous population should be undertaken in the need for and application of simple methods of malaria control, especially larval control.

Ultimately, the problem may be solved by a territory-wide eradication campaign by residual spraying of DDT or a related insecticide. In spite of the enormous difficulties in such areas as the Wahgi Valley with its widely dispersed population, such a campaign could be carried out, and present indications are that the vectors are highly susceptible. As Macdonald (1953) has pointed out, it does not require 100% efficiency in the methods employed to control malaria of this unstable type.

Summary.

There has been little attempt previously to define the upper limits of malaria in the highlands of Papua and New Guinea. For the purpose of the Native Labour Ordinance of the Territory of Papua and New Guinea (1953) an arbitrary limit of 3500 feet (1100 metres) was selected.

Seven species of anophelines, including the three members of the *A. punctulatus* group, occur in the highlands, the latter up to 1900 metres, but their distribution is patchy. These are the only important malaria vectors.

The upper limit of malaria appears to lie between 1800 and 2000 metres, although in some areas such as the Goroka Valley, at 1700 metres, malaria is not endemic. Malaria in the highlands is of the unstable type and epidemics occur from time to time in the marginal areas of its distribution.

There is some evidence that with an increase in European influence in the highlands malaria is extending its boundaries.

The factors which may precipitate an extension of transmission are reviewed. These include the introduction of malaria parasites, an increase in the vector population and climatic changes. Legislation exists to control the treatment of highland labourers entering coastal areas and their subsequent repatriation.

In a period of 20 months in 1950 and 1951 there was a death rate of 4·97 per thousand among highlanders and 5·22 per thousand among coastal natives employed in coastal areas. In spite of the precautions taken to protect highlanders on the coast, many develop clinical attacks while at the repatriation centres. They are given a two-week course of "Camoquin" and primaquine.

Some of the possible natural factors limiting the extension of malaria in the highlands are considered.

Among the control measures suggested are an intensified chemotherapeutic control of highland labourers, the control of foreign natives entering the highlands, the supervision of construction operations which may lead to an increase of vector breeding places, general education of the public and administration personnel and, possibly, at some future date, the inauguration of a malaria eradication campaign by residual spraying of chlorinated hydrocarbon insecticides.

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References.

ANONYMOUS (1943), "Malaria in the South West Pacific", report from the Australian Military Forces Land Headquarters, 1943.

ANONYMOUS (1951), "The Resources of the Territory of Papua and New Guinea", Volume I, Department of National Development, Division of Regional Development, Canberra, A.C.T.

BLACK, R. H. (1954), "A Malaria Survey of the People Living on the Minj River in the Western Highlands of New Guinea", M. J. AUSTRALIA, 2: 782.

CHRISTIAN, S. H. (1947 to 1949), series of typescript reports to the Director, Department of Public Health, Territory of Papua and New Guinea (manuscripts on file in Port Moresby).

CLEMENTS, F. W. (1936), "A Medical Survey in Papua: Report of the First Expedition by the School of Public Health and Tropical Medicine to Papua 1935", M. J. AUSTRALIA, 1: 451.

DE ROOK, H. (1935), in "Report of the 2nd International Pacific Health Conference", Commonwealth Government Printer, Canberra: 23.

EDITORIAL (1956), "Man-Made Malaria", *Papua & New Guinea M. J.*, 1: 116.

EGERTON, W. S. (1954), "An Account of the State of Health and Problems of Medical Interest in Goilala, Papua', M. J. AUSTRALIA, 2: 444.

FREYVOGEL, T. (1956), *"Malaria in tiefer und mittlerer Höhenlage. Untersuchungen in endemischen Gebieten Tanganyikas", Acta tropica*, 13: 58.

GIBLIN, W. E. (1954), "Daraprim in the Wahgi Valley of New Guinea", M. J. AUSTRALIA, 2: 782.

GILLIARD, E. T. (1953), "New Guinea's Rare Birds and Stone Age Men", *National Geographical Magazine*, 103: 421.

GUNTHER, J. T. (1955), "Epidemiology of Malaria in Papua and New Guinea", *Papua & New Guinea M. J.*, 1: 1.

LEE, D. J. (1946), "Records of Anopheles (Diptera, Culicidæ) from High Altitudes of New Guinea", *Australian J. Sc.*, 8: vi.

MACDONALD, G. (1953), "The Analysis of Malaria Epidemics", *Trop. Dis. Bull.*, 50: 871.

METSELAAR, D. (1957*a*), "A Pilot Project of Residual Insecticide Spraying in Netherlands New Guinea, Contribution to the Knowledge of Holoendemic Malaria", University of Leiden, Leiden.

METSELAAR, D. (1957*b*), personal communication.

PETERS, W., and STANDFAST, H. A. (1957), "Report on a Malaria Survey in the Sepik District", M. J. AUSTRALIA, 1: 861.

SIMPSON, C. (1955), "Adam with Plumes", Angus & Robertson, Sydney.

SOUNESS, T. (1957), personal communication.

SPEER, A. (1957), personal communication.

SPENCER, T. E. T., and SPENCER, M. (1955), "Malaria in the Middle Wahgi Valley Area of the New Guinea Highlands", unpublished report to the Director, Department of Public Health, Territory of Papua-New Guinea (manuscript on file in Port Moresby).

SPENCER, T. E. T., and SPENCER, M. (1957), personal communication.

SPENCER, T. E. T., SPENCER, M., JEMBSEN, M. T., and TOMMERUP, J. W. J. (1956), "Special Patrol Report—Malaria in the Mount Hagen Area", *Papua & New Guinea M. J.*, 1: 110.

Territory of Papua and New Guinea (1953), "Native Labour Ordinance 1950-1952, Restrictions on Employment", *Government Gazette*, 36: 296.

CARDIAC ARREST: A PLAN OF ACTION.

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To be completely successful, cardiac massage producing adequate cerebral oxygenation must commence within three minutes of cardiac arrest. It is therefore most necessary that no time should be lost in first recognizing arrest and in then carrying out a methodical plan for its reversal. Much literature concerning the treatment of cardiac arrest now exists, but it is difficult to find such a plan described in a form which can be read step by step by those called upon in this emergency. After reviewing recent literature the following scheme of action has been formulated.

Diagnosis.

Diagnosis must be early. Irreparable cerebral damage occurs after three minutes of complete cerebral anoxia. Diagnostic signs are (i) apnœa; (ii) the colour, which is waxy pale or cyanosed; (iii) the absence of peripheral pulse; (iv) the absence of bleeding in the wound; (v) fully dilated pupils.

A request should be made that the surgeon palpate a major vessel, if available. Time should not be wasted in trying to record a blood pressure.

Treatment.

It should be remembered that an artificial circulation and respiration is being provided until the normal cardiac action returns. The following procedure should be followed:

1. Tilt the table with the patient's head down.
2. Inflate the lungs with 100% oxygen by gas machine, bag and mask, or by mouth-to-mouth insufflation. Do not waste time trying to intubate the larynx; do this later.
3. Attempt to restart the heart by a quick right auricular puncture with a fine three-inch needle through the second right intercostal space.
4. Open the chest in the fourth or fifth intercostal space, from the border of the sternum to the anterior axillary line (avoid the internal mammary artery).
5. Note whether the heart is at standstill or is fibrillating.
6. Commence to squeeze the heart at the rate of 50 to 60 pumps per minute. Do not squeeze with the finger tips; a flabby heart may perforate. The heart should be lifted in the whole hand and compressed against the sternum. Massage, to be adequate, must produce a cartoid pulse at once. Continue massage until a blood pressure of 40 to 60 millimetres of mercury (systolic) can be recorded.
7. Incise the costal cartilages above and below the interspace.
8. Insert a rib spreader.
9. Open the pericardium longitudinally.
10. Intubate the larynx.
11. Relieve the surgeon doing the massage (two surgeons relieving each other at intervals of five minutes should be the rule). Massage must continue between these last five procedures.

Then check to see whether asystole or fibrillation is present.

Asystole or Cardiac Standstill.

If asystole is present the heart is flabby, dark and motionless. Continue pumping until normal contractions are resumed. If these are weak and inadequate, (*a*) stimulate the myocardium by injection into the left auricle of three to 10 millilitres of adrenaline 1/10,000 or 0·3 to 1·0 millilitre of adrenaline 1/1000 in three millilitres of saline, (*b*) compress the aorta distal to the origin of the left subclavian artery, to increase pressure of blood to the coronary arteries and brain.

If there is no normal heart beat after several minutes of artificial circulation, (*a*) inject into the left auricle three to 10 millilitres of adrenaline 1/10,000 or 0·3 to 1·0 millilitre of adrenaline 1/1000 in three millilitres of saline, (*b*) inject into the left auricle three to four millilitres of 10% calcium chloride. This restores tone to the heart muscle and may initiate contraction.

Continue pumping between injections. These injections may have to be repeated, but hope must not be lost until pumping has continued for a long period and whilst the pupils remain contracted.

Fibrillation.

If fibrillation is present, the heart appears as if a series of shimmering small waves is passing over its surface. The most effective way to abolish ventricular fibrillation is by electric defibrillation. This will cause asystole, and regular manual compression of the heart is then undertaken.

If the heart is fibrillating, act as follows. (*a*) Establish artificial circulation and respiration. (*b*) Apply the two electrodes firmly to the heart, one posteriorly to the apex, the other to the anterior aspect of the base of the heart. The electrodes must be well covered with saline pads to prevent burning the heart. (*c*) Give one shock for 0·25 second at 180 volts. If arrest occurs treat as mentioned above. (*d*) If the heart is still fibrillating, massage and give a second similar shock. (*e*) If it is still fibrillating, massage and give five to seven shocks at intervals of 0·5 to 1·0 second.

More than one series of shocks may be necessary, and compression of the heart should be carried out between each set of shocks.

If electrical methods fail, (*a*) inject intracardiac procaine, 10 millilitres of 1% solution; or (*b*) inject five millilitres of 4% potassium chloride into the heart. Both these drugs will cause asystole. Continue pumping. If this asystole persists, inject 0·3 millilitre of one in 1000 adrenaline in three millilitres of saline; or inject three millilitres of one in 10,000 noradrenaline, *plus* three to four millilitres of 10% calcium chloride.

Once the natural circulation is restored:

1. Augment and support it with transfusions and noradrenaline if necessary. In some cases there is considerable depletion of the circulating blood volume; this must be corrected as soon as possible.
2. Secure the bleeding points, especially the internal mammary artery near the sternal border.
3. Suture the pericardium loosely with interrupted sutures.
4. Suture the costal cartilages.
5. Close the chest, with the use of an underwater seal, if the normal heart beat has continued uninterrupted for some minutes.

If artificial circulation is not restored in three to four minutes, cerebral œdema from anoxia will almost certainly occur, and measures must then be taken to prevent this, (i) by intravenous injection of 100 millilitres of 50% sucrose solution (repeat this injection every four hours); (ii) by intravenous injection of 0·1% procaine twice a day.

Summary.

A scheme of action in cases of cardiac arrest is described.

Attention to the details of treatment is essential, and it is a tremendous help if these are well known before such an emergency occurs. The necessary equipment should always be readily available. Treatment will be successful only if it is early and if it is efficient right from the start.

References.

ADAMS, H. D., and HAND, L. V. (1942), "Twenty-Minute Cardiac Arrest with Complete Recovery: Principles of Prevention and Treatment", *J.A.M.A.*, 118: 133.

AMENT, R., PAPPER, E. M., and ROVENSTINE, E. A. (1951), "Cardiac Arrest During Anæsthesia: Review of Cases", *Ann. Surg.*, 134: 220.

ANDERSON, R. M., SCHOCH, W. G., and FAXON, H. H. (1956), "Cardiac Arrest", *New England J. Med.*, 243: 905.

BEECHER, H. K., and TODD, D. P. (1954), "A Study of the Deaths Associated with Anæsthesia and Surgery", *Ann. Surg.*, 140: 2.
BRIGGS, B. D., SHELDON, D. B., and BEECHER, H. K. (1956), "Cardiac Arrest", *J.A.M.A.*, 160: 1429.
EFFLER, D. B., and GROVES, L. K. (1955), "Elective Cardiac Arrest in Open Heart Surgery", *Surgery*, 142: 446.
HELLER, M. L., NYDER, J., and STORRS, R. C. (1955), "Nitrous Oxide-Oxygen-Curare Anesthesia for the Geriatric Patient: An Electrocardiographic Study", *Current Res. in Anes. & Analg.*, 34: 121.
HINCHEY, P. R., and STRAEHLEY, C. J., junior (1952), "Cardiac Arrest in the Operating Room: Report of Five Cases Occurring in a Community Hospital", *New England J. Med.*, 247: 1003.
LEATHAM, A., COOK, P., and DAVIES, J. G. (1956), "External Electric Stimulator for Treatment of Ventricular Stand-still", *Lancet*, 1185: 1189.
ROB, C., and SMITH, R. (1957), "Operative Surgery", Volume 3, Butterworths, London: 168.
SAKLAD, M. (1953), "Inhalation Therapy and Resuscitation", Thomas, Springfield, Ill.
SLOAN, H. E. (1950), "Vagus Nerve in Cardiac Arrest: Effect of Hypercapnia, Hypoxia, and Asphyxia on Reflex Inhibition of Heart", *Surg., Gynec. & Obst.*, 91: 257.
STEPHENSON, H. E., junior, REID, L. C., and HINTON, J. W. (1953), "Some Common Denominators in 1200 Cases of Cardiac Arrest", *Ann. Surg.*, 137: 731.
WYLIE, W. D. (1956), "The Treatment of Cardiac Arrest", *Brit. J. Anaesth.*, 551: 556.

FEMALE URETHRAL STRICTURE.

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STRICTURE of the female urethra is a common condition which is seldom recognized in general practice. It produces much chronic ill-health and discomfort but is easily treated. Although the condition is well known to urologists, a description of its symptomatology and treatment rarely finds its way into standard textbooks or journals.

Reports of Cases.

Before the ætiology, symptoms and treatment of the condition are considered, the following cases are presented as typical of the many which are seen in private urological practice. The majority of the patients had been treated by simple measures for some time before being referred.

CASE I.—The patient, a sixty-year-old woman, complained of frequency of micturition for six months. During this time she had felt very tired and had had numerous night sweats. Her physician had found numerous white cells in her urine, and on culture there was a growth of *Bacillus coli*, which was sensitive to several antibiotics. She had several courses of these drugs, and although the infection improved it was not eradicated and tended to recur once the therapy was withdrawn. Her intravenous pyelogram was within normal limits. All tests for tuberculosis gave negative results. At cystoscopy the external meatus was whitish in appearance, and a 21F cystoscope could not be passed; the urethra would admit only an 18F sound, but was readily dilated to 22F. The bladder was not trabeculated, and there was no residual urine. Ureteric orifices were normal, but the trigone was reddened, and at the bladder neck there were numerous polypi and heaped-up masses of hypertrophic mucosa. After a course of dilatations her urine became free of cells and organisms, and her symptoms disappeared.

This patient illustrates the most common mode of presentation of these cases, that is, a chronic infection which will not clear up with routine therapy. This patient had a mild stricture, and although there was considerable evidence of chronic inflammation at the base of the bladder, there were no signs of obstruction. There was no trabeculation, residual urine or diminished renal function.

CASE II.—The patient, a woman aged 55 years, presented with severe frequency of micturition both by day and by night. There was intermittent dysuria and she had pain in both groins and in the lower part of the back. She also had pain on the front of her thighs. The symptoms had been present for about 14 years, but had become worse during the last two to three years. Treatment by means of urinary antiseptics, although producing remissions at first, were now ineffective. Her urine contained many white cells in a high-power field, and on culture a growth of *B. coli* was obtained. Her intravenous pyelogram was within normal limits, and her blood-urea nitrogen value was 18 milligrammes per 100 millilitres. At cystoscopy her urethra was very narrow and the 21F cystoscope could not be passed. It would admit only a 16F sound, and there were four ounces of residual urine. The bladder was trabeculated, and there was considerable pitting. The trigone was reddened, and there was a marked collar of chronic inflammatory tissue around the bladder neck, projecting into the bladder. After dilatation her symptoms were dramatically improved, and after a short time her residual urine was eliminated.

This patient had a much more severe stricture than the patient in Case I, and cystoscopically her bladder resembled the bladder seen in male obstructive disease with trabeculation and residual urine. Once again there was considerable evidence of chronic inflammation at the bladder neck.

CASE III.—The patient, a woman aged 26 years, had a severe attack of "cystitis" six weeks after a normal delivery. She was treated with the antibiotic specific for her infection, but although the acute symptoms subsided she was still troubled by frequency and dysuria. It is of interest that she had experienced previous attacks of "cystitis", the first while she was on her honeymoon three years before. These attacks, which had resembled the recent attack, had cleared up rapidly with simple therapy. At cystoscopy her urethra would not admit a 21F cystoscope, and only an 18F sound could be passed, but there was no trabeculation or residual urine, although there were several polypi in the posterior urethra and redness around the bladder neck extending onto the trigone. She was greatly improved after dilatation, and her infection rapidly cleared up.

This patient is in a younger age group, and here the exciting factor was parturition. Her original attack, commonly known as "honeymoon cystitis", was probably due to coital trauma, and is a common antecedent to recurrent attack of cystitis in females.

CASE IV.—The patient, a girl aged 20 years, complained of four attacks of pyelitis during the previous 12 months. The attacks consisted of frequency and dysuria, fevers and headache with pain in the right loin. The attacks had responded slowly to treatment. She had had two or three previous attacks, the first when she was 11 years of age. She was also an enuretic, and on this account she was referred for treatment. The bed-wetting was variable, but always worse after an attack of pyelitis. The bed-wetting had not responded to any medical or training regimes. She had no incontinence, and there was no neurological disorder. At cystoscopy the standard 21F cystoscope could not be passed. After an 18F sound was passed her urethra was readily dilated to 24F. She had no residual urine, the bladder was of normal capacity and there was no trabeculation. There was considerable evidence of inflammation at the bladder neck and in the posterior urethra. After a course of dilatations this girl has had only an occasional wet bed and has been free of infection apart from one attack after influenza.

This case in a girl is interesting, as she presented as a bed-wetter, and in her case the symptom has been almost completely eradicated after dilatations.

General Description.

Calibre of the Urethra.

A normal female urethra will readily admit a 24F or 26F sound. A urethra which will admit only a smaller instrument without pain must be considered to be narrower than normal. While the majority of patients will admit a 24F sound readily, it is not uncommon to find an apparently normal urethra which will admit only a smaller instrument, and it must be assumed there is a considerable amount of normal variation in urethral calibre.

Symptoms.

The symptoms are those that follow the infection which is associated with the stricture. Frequency is common, as is dysuria. These infective episodes occur as attacks, but as the condition progresses they become longer and more difficult to treat. There may be hæmaturia, which is usually terminal. These infective episodes may be quite acute,

with fever, headaches and pains in one or other loins suggestive of "pyelitis". Pains in either iliac fossa, suprapubically or in the lower part of the back, are common, while pain in the anterior and medial aspects of the thighs is frequently encountered. General health seems depressed, and irritability and tiredness are frequent. There may be headache and sweats in more severe cases. There is seldom any complaint of difficulty with micturition comparable with the straining found in males; however, many patients do complain of a vague difficulty and feeling of incomplete emptying. Clinical examination reveals little except some occasional tenderness in either iliac fossa.

Investigations.

The urine is found to contain a variable number of white cells. The findings are influenced considerably by the amount and kind of treatment the patient has had. An intravenous pyelogram seldom reveals any abnormality, but must be performed to exclude any upper tract pathological condition which may be causing the chronic infection. Even in severe cases the blood urea nitrogen test or other renal function tests seldom give abnormal findings. At cystoscopy the urethra is calibrated by the passing of either straight urethral sounds, Hegar's dilators, or Clutton's male urethral sounds. These are passed with the use of a local anæsthetic; xylocaine jelly is quite satisfactory. The normal urethra should allow a 24F sound to pass, but to avoid pain in the strictured case it is wise, when calibrating, to begin by passing a 20F or smaller sound, followed successively by a 22F and 24F, and then the cystoscope is passed. Residual urine is measured, the urine collected is sent for culture, and sensitivity tests are performed. The bladder mucosa is inspected. There may be trabeculation with some pitting and small diverticula, but in mild cases there may be no abnormality. The trigone is frequently reddened, and there may be metaplasia, which resembles a pseudo-membrane, present. The bladder neck almost always shows signs of chronic inflammatory changes. Polypi, small granulomatous areas and heaped-up areas of hypertrophied mucosa are all common, while in some cases the bladder neck may be surrounded by a collar of inflammatory tissue, which projects into the bladder. The urethra shows similar changes in its posterior segment.

Age.

Cases are found in all age groups. The majority of patients are over 45 years of age, but many come from the active reproductive span of life. It seems probable that senile vaginitis is commonly an accompanying disease in the older group, while trauma and infection, after parturition and coitus, play a major role in the younger group. Children and adolescents may also be affected. A congenital stricture is described.

Ætiological Factors.

In the older literature much prominence is given to the influence of the gonococcus in causing these lesions. However, this is now extremely uncommon. It does seem possible, in those women who have a smaller urethra, that infection once introduced is less readily eliminated, and the infective process causes in turn further fibrosis and narrowing. On the other hand, it may be that the trauma and infection alone cause all the urethral narrowing due to œdema and fibrosis. Certainly senile vaginitis, parturition and coitus are important predisposing factors. Some cases have been seen post-operatively, possibly due to catheter trauma, and in one case a tight anterior colporraphy resulted in a narrowing of the urethra. In a minority of cases the narrowing has been at the terminal or anterior urethra, and these are in the main severe strictures, but in the majority the stricture is in the posterior segment, and these are milder in degree.

Treatment.

Treatment is easily carried out by simple dilatation of the urethra, using straight urethral sounds, Hegar's dilators or Clutton's male urethral sounds. At first the dilatations are performed every four weeks, but after three or four treatments it may be possible to lengthen the period between dilatations until a dilatation every six months or so is all that is required. The aim should be to achieve a urethra which will allow the easy passage of a 24F or 26F sound. During the initial dilatations it is wise to give the patient a course of the specific antibiotic indicated by the culture and to follow it up with one of the soluble sulphonamides. After all dilatations it is wise to give the patient one of the soluble sulphonamides for three to five days.

The response to a dilatation is diagnostic of the condition. It accounts for the apparent paradox of a patient being benefited by a cystoscopy. If a urethral stricture is indeed the causative lesion there will be considerable, often dramatic, improvement after dilatation. The symptoms will disappear, and after a short course of dilatations the cells will disappear from the urine, which will become sterile. Symptoms tend to recur after a time. At first this takes about three weeks, but after a time it may be many weeks before symptoms return. This response is the guide when the decision is made that the periodicity of the dilatations can be lengthened.

Summary.

Female urethral stricture is a common condition. It is inadequately described in standard textbooks. Four representative cases are described, and a general description of the syndrome is given, including treatment.

LOCALIZED ŒSOPHAGITIS DUE TO DRUGS.

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IT is generally recognized that localized gastritis can be produced by drugs, especially aspirin (Hurst and Lintott, 1939; Douthwaite, 1938). Medicinally induced œsophagitis, though essentially similar, has received scant attention. Unless the characteristic syndrome is known, it may be mistaken for a more serious disease. Five cases seen within the past six years are recorded.

Reports of Cases.

CASE I.—A physician, aged 33 years, swallowed a three-grain capsule of "Sodium Amytal" without water. Four minutes later there was slight retrosternal discomfort which disappeared immediately after drinking. On awakening next morning, he felt a mild burning pain localized to an area one and a half inches in diameter beneath the sternum at the level of the second costal cartilage. Food was swallowed normally without effect, but the condition was aggravated by very hot tea. The pain increased over the next four hours, and there was a sense of something pressing on the sternum; it did not radiate and was unaffected by movement or respiration. There was no local tenderness. The general condition was unaffected. There were no electrocardiographic changes, and a barium swallow passed normally. The barium gave some relief. Small repeated doses of liquid paraffin produced considerable improvement, as also did cream and olive oil. The pain persisted for 24 hours, then gradually subsided over the next two days. At any time during this period an acute exacerbation could be produced by drinking very hot tea or diluted vinegar. Six years later the patient was well, with normal electrocardiographic findings.

CASE II.—A man, aged 36 years, retained a capsule of "Sodium Amytal" in the mouth for two minutes before washing it down with water. The next day he reported a localized burning substernal pain with definite sense of something pressing on the sternum; he did not associate this with the taking of the capsule on the previous night. A barium swallow passed normally; electrocardiographic findings were within normal limits. Pain was much worse after taking hot drinks but relieved by olive oil. Pain disappeared after two days, and the patient has remained well for two years.

CASE III.—A woman, aged 23 years, took an 0·25 gramme capsule of "Chloromycetin" and washed it down with hot tea. Next day she complained of a severe, burning, constricting pain confined to an area one inch in diameter just above the suprasternal notch. It was made worse by hot or sour fluids

and very much worse when she attempted to eat curry. There was considerable immediate relief from small repeated doses of liquid paraffin. Pain disappeared after 48 hours. The patient did not associate the onset of the pain with the capsule taken on the previous night. She is alive and well four years later.

CASE IV.—A woman, aged 26 years, took two 50-milligramme capsules of "Benadryl" for nausea of pregnancy. She held them in her mouth for some little time and finally washed them down with hot tea. Three hours later localized burning pain commenced over an area one inch in diameter at the level of the second costal cartilage; the pain was made worse by hot drinks but greately relieved by olive oil. The patient did not mention the taking of the capsules until specifically questioned. The pain disappeared after two days, and the patient is well three years later.

CASE V.—A physician, aged 45 years, was disturbed while eating aspirins in his consulting room; he hurriedly swallowed a whole tablet without water. The incident was forgotten. Four hours later he experienced a sense of uneasiness beneath the sternum which gradually increased and became a burning constricting pain localized to an area one inch in diameter just to the left of the midline at the level of the second costal cartilage. Pain was unaffected by coughing or exertion, and nitrites gave no relief. The condition was unaffected by food and greatly worsened by very hot tea. The ingestion of cream and butter gave rapid relief. The pain disappeared after 12 hours and has not recurred.

Endoscopy was not thought justifiable in any of these cases. The aggravation of pain by hot or acid fluids and its invariable relief by oil or cream were considered proof of a lesion in the œsophageal mucosa. The site of pain was what would be expected from a lesion at the point where the gullet is constricted by the left bronchus, or, in one female case, the post-cricoid region.

Comment.

These cases have the following interesting features in common, and would appear to manifest a definite syndrome, which if not recognized may lead to a suspicion of coronary occlusion: (i) The interval of three to 12 hours between medication and the onset of symptoms. Only in the first case did the patient realize that there might be any connexion. The diagnosis will be missed unless the patient is closely questioned. (ii) The strictly localized burning pain, without skin tenderness, which was unaffected by respiration. Three of the five patients spontaneously used the expression "something pressing". (iii) The aggravation of the pain by hot or sour fluids, and the immediate relief from oil or cream. This test is diagnostic. (iv) The hasty or ill-advised swallowing of a capsule containing powder. All but one of the cases were due to this.

No similar cases were seen in a large hospital in-patient population over the same period. Presumably these people take capsules in a proper manner and wash them down with cold water.

Tests show that the modern capsule is excellent when swallowed immediately and washed down with cold fluid. If held in the mouth for two minutes or placed in hot tea or coffee, it forms an intensely sticky mass of capsule and contents mingled together. It can be well imagined that if this stuck to the œsophageal wall it could produce considerable local inflammation.

The passage of similar capsules filled with barium was observed on the X-ray screen. Those swallowed with cold water passed immediately into the stomach; those swallowed with hot tea travelled slowly and hesitantly. One capsule which was swallowed with hot tea, after being held in the mouth for a minute, became stuck two inches above the cardia. It remained there for six minutes and then gradually broke up, but a small blob of barium remained visible for a further five minutes.

Summary.

1. Five cases of localized retrosternal pain, presumed to be due to drug-induced œsophagitis, are recorded.

2. The typical nature of the pain, its relief by oil and aggravation by hot drinks, is described.

3. The delay of three to 12 hours between the taking of the drug and the onset of symptoms, so that the patient does not realize the connexion, is stressed.

4. Some experiments with capsules are described.

5. Certain unsatisfactory features of the modern capsule are noted, and the suggestion is made that manufacturers should stress that they be swallowed immediately and washed down with cold water.

Acknowledgement.

I wish to thank Dr. Pattinson, radiologist to the Middlesex Hospital, London, who carried out the X-ray examinations.

References.

DOUTHWAITE, A. H. (1938), "Recent Advances in Diagnosis and Treatment", *Brit. M. J.*, 1: 1143.

DOUTHWAITE, A. H., and LINTOTT, G. A. M. (1938), "Effects of Aspirin and Certain Other Substances on the Stomach", *Lancet*, 2: 1222.

HURST, A., and LINTOTT, G. A. M. (1939), "Aspirin as a Cause of Hæmatemesis: A Clinical and Gastroscopic Study", *Guy's Hosp. Rep.*, 89: 173.

THE PYLORIC CANAL, URINARY BLADDER AND PERINEUM OF NEW-BORN INFANTS.

By J. B. CLELAND,

Emeritus Professor of Pathology, University of Adelaide.

THE pyloric canal in the new-born infant is a very definite anatomical structure, clearly defined both externally and internally from the fundus above and very sharply demarcated from the duodenum below. On incising it, the thick muscle appears as a cartilaginous-looking layer attenuating upwards. In so-called "congenital stenosis" there is merely an exaggeration of this appearance. The inner aspect is sometimes longitudinally rugose. In 14 consecutive cases in which it was measured, the lengths in centimetres (arranged in order) were 1·1, 1·2(4), 1·6(2), 1·7, 1·8(2), 2, 2·2, 3·1 and 3·7. The infant in whom it was 1·7 centimetres when contracted (this specimen could be stretched to 2·5 centimetres) weighed eight pounds seven ounces. The infant in whom it was 3·1 centimetres weighed seven pounds two ounces, but the one with a canal of 3·7 centimetres weighed only four pounds nine ounces. It was 1·2 centimetres long in an infant who weighed eight pounds twelve ounces and 2·2 centimetres in another weighing eight pounds two ounces. It is evident that the length does not necessarily vary with the weight.

Why should the pyloric canal be so muscular at birth? I suggest that it is to enable the mucus and cell-debris that collect in the stomach before the baby is born to be passed on into the intestines against the considerable pressure of the amniotic fluid. There it would contribute its quota to the meconium.

The neonatal urinary bladder is surprisingly muscular. I suggest that here again the muscle is proportionately thick, compared with adults, to enable the bladder to be emptied against the high intra-uterine pressure.

The perineum of infants when the legs are slightly separated is usually nearly a plane surface, though a sulcus of varying depth may be present. An extreme example of the latter was seen in a female infant weighing eight pounds one ounce. In place of the perineum there was a cleft 1·5 centimetres deep, at the bottom of which was a septum 1·5 centimetres broad separating the vagina from the rectum. The anal cleft extended unusually high so as to reach nearly to the level of the iliac crests.

Other interesting features of the new-born are that the subcutaneous fat is lobulated, the cervical os is transversely rugose and ragged-looking, the cervical portion of the uterus is longer than the body, the epiglottis is usually folded (this is occasionally seen in adults), and the medulla and pons are firm and white (medullated) while the rest of the brain is almost gelatinous.

Reports of Cases.

RECTAL BLEEDING DUE TO OVARIAN CYST.

By BARTON VENNER, ROLAND BEARD, JUNIOR, AND V. J. ODLUM,
Adelaide.

RECTAL BLEEDING is a symptom whose value is that of the red flag for danger, and calls for investigation and for caution. It is true that a carefully taken clinical history and an examination may suggest the likely diagnosis or even supply a satisfactory one, but one must be certain that it is the right one. For this purpose the appropriate endoscopic and radiological investigations must be considered and rejected only after deliberation.

The case to be described illustrates the difficulty of securing an exact pre-operative diagnosis.

Clinical Record.

The patient, aged 33 years, a married woman with two children, was found on routine abdominal examination to have a round tumour some five inches in diameter, lying in the left iliac fossa. On questioning, she gave the history that for about nine years she had passed blood per rectum intermittently, and that she had had colicky lower-abdominal pain and the desire to empty her bowel. At these times she would, in fact, pass only blood. The colon was radiologically examined at this time, without a diagnosis being made.

The attacks had continued intermittently, but she had remained well, and had had two children. During the second confinement, three years before, delivery was delayed a good while, but then occurred rapidly when Cæsarean section was being considered. She was subject to some intermittent discomfort from "wind" in the stomach, until during recent weeks, when the bleeding and colicky spasms became more severe. A good deal of blood was passed, with tenesmus, but at times the motions were normal. Her appetite, weight and health generally were normal. There were no significant previous illnesses.

On examination, a rounded tumour, five inches in diameter, was palpable in the left lower abdomen. It was probably cystic in nature, smoothly rounded, non-tender, and not in the abdominal wall. It was freely mobile from side to side, but not downwards, and could not be pushed into the pelvis. Bimanual examination revealed a uterus of normal size and without apparent connexion to the abdominal mass. The ovaries were not felt.

On sigmoidoscopic examination, dark blood unmixed with fæces was observed to be descending from above at six inches; beyond this point the sigmoidoscope could not be passed. There were hæmorrhoids of first degree, which were not bleeding. A barium enema X-ray examination showed no intrinsic abnormality of the large bowel. However, the sigmoid colon was displaced in a graceful curve to the right, apparently by a tumour situated to the left of, or in, its mesenteric attachment.

A diagnosis of benign cyst or tumour of the sigmoid mesentery was made. At laparotomy, through a lower midline incision, a large benign ovarian cyst of ovoid shape was found nestled in the left iliac fossa, and lying to the left of and in front of a rather long sigmoid mesentery. There was no attachment to the colon or rectum, the whole of which appeared to be normal. The pelvic brim was well rounded, with very little protuberance of the sacral promontory. The cyst appeared to be just too large to engage in the pelvis. The elongated pedicle was easily secured to remove the cyst, which was serous in type.

Discussion.

It was easy to visualize how hæmorrhage into the colon might be produced in this patient. It appeared that the sigmoid arteries and veins were sharply folded back on themselves in the root of the long meso-sigmoid, and compressed there between the cyst and the pelvic brim. No visible engorgement or other pathological change was present, but it is suggested that recurrent venous engorgement was responsible both for the griping pain and for the bleeding of which this patient complained.

The removal of this cyst had resulted in complete relief of the pain and hæmorrhage up to the time of writing, five months later.

UNUSUAL COMPLICATION OF ELECTRO-SHOCK THERAPY.

By MARY ALLEN HIELD, M.B., Ch.B., D.P.H., D.R.C.O.G.,
Mental Hospital, Callan Park, Sydney.

ON August 14, 1957, a frail, elderly woman, aged 63 years, was admitted to Callan Park Mental Hospital, Sydney. There was a history of marked loss of weight and occasional attacks of flatulence. Psychiatric examination revealed severe depression with agitation, aural hallucinations and strong feelings of guilt. Similar symptoms had first occurred three years previously; the patient had received treatment in a private hospital, first as an in-patient and later as an out-patient, by electro-shock therapy, the last treatment having been given in September, 1956. Although considerable improvement had occurred in the patient's condition after treatment, her condition had gradually deteriorated until her admission to Callan Park. After her admission to hospital the patient was placed on a special diet and was hand fed, but owing to the severity of her symptoms shock therapy was commenced on August 27, 1957, using the "Minecta" apparatus and relaxants, namely "Brevidil" and pentothal given intravenously. After the third shock treatment on September 3, the patient collapsed and respiration was slow to reestablish itself, but commenced satisfactorily as soon as an airway was inserted. The heart beat was at first slow and regular, but the patient appeared shocked and palpation of the abdomen revealed distension and a hard, board-like rigidity. The patient complained of severe abdominal pain, and a rectal tube and a Ryle's stomach tube were inserted, the latter causing some eructations but no flatus was passed.

As the patient's general condition deteriorated rapidly, the blood pressure falling from a normal 170/105 millimetres of mercury to 85/55 millimetres of mercury, the consulting physician and acting medical superintendent of the hospital were called in consultation and a nor-adrenaline intravenous transfusion was commenced almost immediately.

The blood pressure slowly rose until some seven hours later, when it reached 130/70 millimetres of mercury, but it was necessary to continue the nor-adrenaline transfusion for another 24 hours.

The patient still complained of abdominal pain and was taking only sips of fluid by mouth, but her mental condition appeared quite normal. Two days later her condition appeared to be deteriorating and the intravenous transfusion of 4% glucose was commenced.

Aspiration of stomach contents resulted in the withdrawal of 360 millilitres of dark green fluid, and after this smaller quantities were withdrawn at hourly intervals for 24 hours. The following day the patient was transferred to Royal North Shore Hospital, Sydney, with a tentative diagnosis of ruptured peptic ulcer.

On arrival at the hospital the patient's condition was reported as moderately satisfactory; she had had no bowel action, her pulse rate was 86 per minute, and her blood pressure was 140/70 millimetres of mercury, with no dehydration, the abdomen being resonant to percussion and the liver dullness absent.

X-ray examination of the abdomen showed free gas present, some distension of the small bowel and a fluid level present in the ascending colon. Laboratory investigations gave the following values for serum electrolytes: sodium, 138 milligrammes per litre; potassium, 3·4 milli-

grammes per litre; chlorides, 95 milligrammes per litre; bicarbonate, 25 milligrammes per litre. The serum amylase was normal. Blood urea content was 23 milligrammes per 100 millilitres. The hæmoglobin value was 13·6 grammes per 100 millilitres, the white cell count 7800 per cubic millimetre, and the erythrocyte sedimentation rate eight millimetres per hour. Treatment was instituted with penicillin and streptomycin, a Meulengracht diet and the administration after some days of "Amphojel", two drachms every two hours, and tincture of belladonna, 15 minims three times a day and 20 minims at night.

There was a steady improvement in the patient's condition, distension subsided, the bowel movements became regular and pain disappeared. The patient was returned to Callan Park on September 23 in excellent physical condition, but her symptoms of depression had recurred. Since then she has been treated with "Largactil" and "Ritalin" and has shown considerable mental improvement, sufficient to have painted the background scenery for a puppet show organized recently by the occupational therapists.

Comment.

There appear to be few cases reported in the literature of rupture of a peptic ulcer after electro-shock therapy.

According to Selye (1951), in patients undergoing ACTH therapy, intense abdominal pain and exacerbation of pre-existing peptic ulcers may occur, presumably because ACTH stimulates the production of pepsin, gastric hydrochloric acid and the total amount of gastric juice.

Electro-shock therapy, which is a potent stress-producing factor, increases the patient's production of ACTH. Early *et alii* (1951) has reported that one electro-shock therapy has a greater action than 50 milligrammes of standard ACTH preparation. In patients with pre-existing duodenal ulcer this increase may be sufficient to cause a perforation of the ulcer.

Naomi Raskin and William Hull (1954) report the case of a white woman, aged 31 years, admitted to Boston State Hospital who died after her thirty-fourth shock treatment. Although she died of carcinoma of the lung, autopsy also revealed a duodenal ulcer which had perforated, with peritoneal reaction but without peritonitis because of the protective adhesion of the omentum.

The Hon. W. S. Maclay in his presidential address to the Royal Society of Medicine in 1952 quotes amongst his 62 cases of death after electro-shock therapy only one due to ruptured intestine and peritonitis, this occurring in an aggressive schizophrenic, aged 28 years, with a late onset 48 hours after treatment.

It is of interest to speculate on the many causes which could give rise to peptic ulceration and perforation. In 1932 Cushing, in a paper entitled "Peptic Ulcer and the Interbrain", described 11 cases of interbrain lesions, three of which ended in ulcer perforation. Cushing postulated that erosions and ulcers in the gastro-intestinal tract are not infrequently associated with cerebral lesions and post-cranial operation periods.

Acknowledgements.

I wish to thank the Inspector-General of Mental Hospitals and the medical superintendent of Callan Park Mental Hospital for permission to report this case. I also wish to thank Dr. S. Sandes and Dr. G. S. Nagy for their immediate help and advice concerning treatment, and Miss J. Hunter of the Public Health Library for her help with the references.

References.

CUSHING, H. (1932), "Peptic Ulcer and the Interbrain", *Surg., Gynec. & Obstet.*, 55: 1.

EARLY, D. F. M., *et alii* (1951), "Use of ACTH in Psychiatry: (*a*) Response Tests, (*b*) Therapy, (*c*) Physiological Observations and Comparison with Electro-Convulsive Therapy", *J. Endocrinol. (Proc. Soc. for Endocrinol.)*, 7: 18.

MACLAY, W. S. (1953), "Death due to Treatment", *Proc. Roy. Soc. Med.*, 46: 13.

RASKIN, N., and HULL, W. M. (1954), "Rapidly Growing Carcinoma of the Lung and Perforated Duodenal Ulcer in a Patient Treated with Electric Shock", *J. Nerv. & Ment. Dis.*, 119: 449.

SELYE, H. (1951), Annual Report on Stress, Montreal.

Reviews.

Psychoprophylactic Preparation for Painless Childbirth. By Isidore Bonstein, M.D.; 1958. London: William Heinemann (Medical Books), Limited. 8″ x 5″, pp. 144, with illustrations. Price: 12s. 6d. (English).

IN this book Dr. Bonstein presents the theory and practical approach to a method whereby, through the education, mental and physical, of pregnant women, painless childbirth is achieved. This method, "based on the theory of superior nervous activity, established by Pavlov", originated with Dr. Fernand Lamaze, in whose hospital in Paris, of over seven thousand prepared women, no less than two-thirds achieved good to excellent results and only 5% were failures. The introductory chapters include discussion of Pavlov's work on conditioned reflexes, psycho-physiology and the study of pain. Then follows the course of eight lectures as given to patient-groups during the third trimester. These are comprehensive, but presented in a simple conversational way. They discuss neurology, the theory behind the method, the physiology of respiration, neuromuscular education and relaxation, the detailed events of labour, and finally the behaviour to be followed during the various stages. Specific exercises are gradually introduced, so that the patient becomes muscle and diaphragm conscious, can relax at will, recognizes her Braxton Hicks contractions and automatically accompanies them with fast shallow breathing, and learns the meaning of "bearing down". Patients are shown the delivery room, the special pillows used during labour, the oxygen apparatus which is used in the second half of labour as an essential part of the method, and films of painless childbirth. The book ends with reports written by four enthusiastic American mothers who experienced painless childbirth after Lamaze's preparation.

This method has nothing in common with hypnotism. The preparation aims at the establishment of special, somewhat unstable conditioned reflexes, and success may and must be obtained by activation of the cerebral cortex, not by depression and inhibition. "Reorganization" of the patient's cortical activity results in the suppression of the conditioned pain reflexes (said to be a product mainly of ignorance about childbirth) and the creation of "new useful conditioned reflexes capable of associating uterine contraction with a strong, positive and painless activity". Serious preparation is necessary; time and patience are demanded. The maintenance of the conditioning throughout labour requires psychology on the part of all concerned, from the hall porter to the *accoucheur*. True teamwork is essential. The application of this method appears something of a tall order, but given the necessary "set-up"—for example, a cottage hospital and an enthusiastic staff—there is no reason why results in Australian women should not outstrip those in the hospital of its origin.

The Mammalian Cerebral Cortex. By B. Delisle Burns, B.A., M.R.C.S., L.R.C.P.; 1958. London: Edward Arnold, Limited. 8¾″ x 5″, pp. 128, with illustrations. Price: 21s. (English).

IN this little volume Professor B. Delisle Burns attempts to elucidate some of the functions of the cerebral cortex in mammals, more specifically the cat. The experimental isolate is a "slab" of cortex from the suprasylvian gyrus about twenty millimetres by five millimetres in area and undercut at a depth of about four millimetres. The slab is left *in situ* in the decerebrate cat, and its blood supply is damaged as little as possible; but there is no mention of histological check to assess any retrograde damage to neurons, particularly in chronic preparations. Stimulation of normal cortex produces a surface negative response, which is little affected by anæsthesia. Stimulation of the isolated cortical slabs also produces a surface negative response; but if stimulation strength is increased, this changes to a prolonged burst of neural activity, in which parts of the cortex become electropositive, and this burst spreads throughout the cortical slab. The electropositive response is never seen in intact cortex and is readily suppressed by anæsthetics. Moreover, the completely isolated cortex lacks the spontaneous activity of normal cortex. Burns attributes the surface negative response to a discharge from cells which, from his diagram, appear to be in lamina V, and the surface positive response to cells of about lamina III. The surface negative response spreads superficially, probably in lamina I, at some two metres per second, but attenuates rapidly and is lost ten millimetres from the point of stimulation. The surface positive response, however, spreads without attenuation to the borders of the cortical strip and may persist for up to five seconds. This is considered the response of a functional unit—a group of cells—and, despite the artificial conditions,

the author believes that the surface positive type of response must be a component of normal behaviour in the intact brain. The work continues with a detailed consideration of after discharges, spontaneous activity in central neurons and the transmission of excitation within the cerebral cortex. In each case the discussion is interesting, though necessarily inconclusive. This is particularly so in regard to the spreading cortical depression which follows repeated stimulation of any cortical point. The wave of depression spreads concentrically at about 0·5 millimetre per second and abolishes any excitability for about one minute. Such depression can be obtained in the isolated slab as well as in the intact brain. It is tentatively ascribed to a spreading potassium leakage. Burns likens this to the phenomenon of cerebral concussion. In the last chapter the author reviews theories of memory and learning. His own suggestion that they may be partly hormonal and partly due to the replacement of old cells by new ones is suggestive but not yet very convincing. The book is essentially a monograph for specialists in this field, but anyone with a reasonable acquaintance with neurophysiology can read it with interest.

Suspension Therapy in Rehabilitation. By Margaret Hollis, M.C.S.P., and Margaret H. S. Roper, M.C.S.P.; 1958. London: Baillière, Tindall and Cox. 8½" × 5½", pp. 232, with 112 illustrations. Price: 25s. (English).

WRITTEN for the most part by physiotherapists for physiotherapists, this book "continues the invaluable pioneer work of Olive Frances Guthrie Smith". The book is divided into two parts: Part I, chapters 1 to 12 (124 pages), deals with the mechanical principles, techniques and methods of suspension, spring and pulley therapy; Part II, chapters 13 to 18 (89 pages), describes the clinical application of suspension spring and pulley therapy to special conditions. The Guthrie Smith suspension apparatus and its principles are very widely used in England, and the apparatus (or adaptions of it) is being seen with increasing frequency in physiotherapy departments in Australia. The apparatus and its principles are invaluable in muscle reeducation and strengthening and in joint mobilization, and hence are very useful in medical rehabilitation. The chief virtue of this book is that it sets out the proper way in which the apparatus and all suspension therapy should be used and the principles on which it depends. Although the sections on physics are a curious mixture of unnecessary detail and confusing brevity, the rest of Part I, dealing with techniques and their application to various parts of the body, is well set out and well illustrated by clear line drawings supplemented by a number of plates. This part should prove invaluable to physiotherapists required to use suspension therapy.

Part II, purporting to deal with the application of suspension therapy to specific conditions, is patchy. The chapters with medical men as authors or co-authors are clearly superior to those written by physiotherapists alone. The chapter on amputations by E. S. Thomson, M.Ch., is a particularly fine one, whereas the chapter on orthopædic conditions by Miss J. A. Huggan needs the authority of an orthopædic surgeon. The chapter on paraplegia by Dr. L. Guttmann and Mrs. D. T. Bell is a masterly exposition of the modern treatment of paraplegia, mainly one suspects by Dr. Guttmann. Suspension therapy, however, gets very brief mention.

In spite of the shortcomings of Part II, the book is a valuable guide to physiotherapists in the correct use of suspension therapy. It should also be read by all doctors who are responsible for supervising physiotherapy departments and by those who commonly prescribe physiotherapy.

Electron Microscopic Atlas of Normal and Leukemic Human Blood. By Frank N. Low, Ph.D., and James A. Freeman; 1958. New York, Toronto, London: McGraw-Hill Book Company, Incorporated. 9¾" × 7", pp. 362, with many illustrations. Price: $25.00.

THIS is the first atlas of its type which gives the ultrastructure of normal and leukæmic peripheral blood cells. The book is well produced and contains numerous illustrations, as its name indicates, and the text is interestingly presented. An account of the technique of preparation of material is an entertaining and useful introduction to the subject.

The first and largest section is devoted to the electron microscopic cellular morphology of normal human peripheral leucocytes and platelets. The authors have selected a large number of photomicrographs and have arranged these systematically so as to present concisely and comprehensively the salient features of the abundant cytoplasmic and nuclear detail as viewed at magnifications of 10,000 to 70,000. This section is entirely descriptive, and most attention has been paid to identification of cell types by means of readily recognizable structures; the terminology of these is extremely simple and is comparable with that associated with the light microscope. In addition, a representative collection of light photomicrographs of peripheral blood smears has been included for comparison.

The subsequent sections deal with leukæmic cells of the various types—namely, myeloid, stem cell, lymphocytic and monocytic. In all these groups excellent descriptions are given of the morphology of the various cells found in the peripheral blood, and these also are illustrated by numerous micrographs arranged in the same systematic, concise and comprehensive manner as for normal blood. Also, in this section, the authors have attempted to cover the development of the granular series by studying the morphology of the primitive cells present in the peripheral blood of patients with acute and chronic leukæmia. Perhaps marrow blood from healthy people would have served a better purpose for this type of study, since we know from histochemical studies and other experimental work that these leukæmic cells, particularly those of acute leukæmia, possess a gross morphological difference from their normal counterparts. However, the problem of localization of material at the magnifications employed is recognized by all who have used the electron microscope; so that, for the time being, this approach must be acceptable.

Since there is no comparable book on this subject, it should serve as a stimulus for further systematic evaluation of the host of findings seen in electron microscopy. Even though the techniques used to make these studies may become obsolete in a few years, this book will still serve as a good reference. It helps to clarify the doubts and arguments as to the nature of the organelles which are vaguely seen in light microscopy, and observations on the platelets give some idea of the possible (and probable) advances of the next few years. This atlas will be found a most valuable addition to the hæmatologist's library.

Anatomy and Physiology for Nurses. By W. P. Gowland, M.D., F.R.C.S., and John Cairney, D.Sc., M.D., F.R.A.C.S.; Fifth Edition; 1958. New Zealand: N. M. Peryer, Limited. 8½" × 5½", pp. 536, with 204 illustrations (including several in colour). Price: 45s. (New Zealand).

THIS is an excellently written and arranged book, and the printing and paper make it a pleasure to read. It should prove a boon to the tired trainee nurse for study and as a reference book. Its scope is quite comprehensive, but each fact is described in such a clear, concise and simple manner that the subject matter can be easily understood and assimilated. The many excellent illustrations will help greatly in the understanding and memorizing of the context. The book's application of anatomy and physiology to medicine and surgery, as seen in the wards, is a definite advantage. The study of anatomy and physiology is made interesting and easy, and the book can be highly recommended as a text-book and reference book for Australian nurses.

The Medical World of the Eighteenth Century. By Lester S. King, M.D.; 1958. Chicago: The University of Chicago Press. 8½" × 5", pp. 368, with illustrations. Price: $5.75.

LESTER S. KING, professor of clinical pathology at the University of Illinois, Chicago, has made a valuable contribution to medical literature and history by writing a number of essays, each reflecting some aspect of the healing art as understood and practised by physicians, apothecaries and charlatans of the eighteenth century. This fascinating period, as Dr. Ilsa Veith observes in an illuminating foreword, had been distinguished by J. H. Baas as "the most important century in the history of culture and particularly the sciences", while Pagel attributed its phenomenal march of progress to "emancipation from religious and political domination, when science could flourish, unhampered by any external pressure".

A great deal of hard work has been undertaken to gather material illustrative of the complicated theories about the human body and its reactions to disease as conceived by famous teachers like Hermann Boerhaave, of Leiden, whose authority was enough for them to be accepted and followed implicitly by most European and American practitioners of the time. In fact, nearly half the book is taken up with the meticulous recital of these discarded medical doctrines, which, were it not for the writer's exceptional literary taste and fluent style, might well prove a strain on the patience and application of the average reader. Hard-pressed graduates of the modern school seem to lack enthusiasm for medical speculations of ancient vintage, especially when administered to them in excessively large doses. However, the chapters which treat of the manners, customs and

behaviour of qualified practitioners, quacks and empirics in dealings with their impressionable patients are entertaining and instructive to read. Each essay is carefully documented with bibliographical references towards the end of the book.

In the preface, which almost amounts to an essay in itself, Dr. King stresses the prime necessity for historians to set about trying to capture the spirit of the past before attempting creative work bearing on the history of any given epoch; in the course of his remarks he discloses a preference for noting similarities rather than differences in human tendencies as compared with those in evidence at the present day. He finds that "in the eighteenth century doctors were just as clever as they are today, just as perceptive and clear-thinking, exerting powers of observation and inference just as keen. At the same time, eighteenth-century physicians were quite as muddle-headed, obtuse, grasping, prejudiced, and contentious as their descendants." His observations on the philosophy of history are sound in conception and eloquently expressed; while the comparisons drawn between the attitudes of the historical novelist and the painter, the true historian and the photographer, are not only ingenious but worthy of contemplation as a study of different artistic methods and techniques.

The Early Diagnosis of the Acute Abdomen. By Sir Zachary Cope, B.A., M.D., M.S. (Lond.), F.R.C.S. (Eng.); Eleventh Edition; 1957. London, New York and Toronto: Oxford University Press. 8¼" × 5½", pp. 204, with 36 illustrations. Price: 29s. 9d.

OVER the years since its first appearance in 1921, this little book has earned well-merited popularity, and has proved a most helpful source of reference, particularly for those in general practice and for resident medical officers. This new edition will enhance that reputation, and those engaged in the teaching of students can draw with assurance from the wealth of information and experience in its pages. The author has made a complete revision of his former work and has made some additions which have become necessary through the advances made in surgical knowledge. In the section on appendicitis, more emphasis might have been made on the difficulties presented by non-tuberculous mesenteric lymphadenitis and the occasional case of torsion of the testis or its appendages; but such criticism is hardly justified when so much has been packed into a small volume with so little evidence of cramming. The text drawings are particularly clear, and the radiographs are classical examples beautifully reproduced. This book can be commended in the highest terms.

Reflexogenic Areas of the Cardiovascular System. By C. Heymans, M.D., and E. Neil, M.D., D.Sc.; 1958. London: J. and A. Churchill, Limited. 9¾" × 7", pp. 272, with 89 illustrations. Price: 56s. (English).

THIS is a very valuable reference book for anyone interested in the reflex control of the circulation. The book discusses mainly the physiology of the baroreceptors and chemoreceptors, and deals briefly with some other reflexogenic zones. Recent developments in electrophysiology have allowed the presentation of new data dealing with the nature of the information that is conveyed from these reflex zones to the central nervous system. The part of the book dealing with the anatomy and electrophysiology of the afferent reflex arcs is particularly well presented. The effector side of the reflex arc is dealt with much more on the lines of Heymans's original monograph; most of the discussion is concerned with the effects of reflex stimuli on the arterial pressure, heart rate and respiration. Only four pages are devoted to the problem of baroreceptor reflexes and cardiac output regulation. The authors discuss the lack of adequate experimental data dealing with the problem of reflex blood-flow redistribution and also stress certain difficulties up to date in the experimental approach to the question of reflex venomotor adjustments.

Unwillingness to discuss these problems has given the book a somewhat disjointed format. Reflexogenic zones are discussed individually, and the problem of central nervous integration is rather avoided. The book includes a useful chapter dealing with reflexes arising from the heart and lungs: the account in this section is largely anatomical, but there is a brief discussion of the important question of atrial volume receptors. The relegation of the Bainbridge and McDowall reflex to "reflexes of uncertain origin" foreshadows a welcome omission of these reflexes from standard texts of physiology, since their role in the intact circulation is dubious. An extensive list of references covers the English and Continental literature. The book is likely to appeal both to medical undergraduates and to practitioners interested in the circulation.

Books Received.

[The mention of a book in this column does not imply that no review will appear in a subsequent issue.]

"What You Can Do for Angina Pectoris and Coronary Occlusion", by Peter J. Steincrohn, M.D., F.A.C.P.; 1958. Sydney, London, Melbourne, Wellington: Angus and Robertson. 7¼" × 4½", pp. 184. Price: 25s.

Written for the layman.

"Principles of Research in Biology and Medicine", by Dwight J. Ingle, B.S., M.S., Ph.D.; 1958. Philadelphia, Montreal: J. B. Lippincott Company. Sydney: Angus and Robertson, Limited. 9" × 5¾". Price: 52s. 3d.

Written primarily for students preparing for or beginning research in macrobiology and medicine.

"The Long Road Back: The Story of My Encounter with Polio", by Edward Le Comte; 1958. London: Victor Gollancz, Limited. 7¾" × 4½", pp. 192. Price: 20s. 6d.

The Associate Professor of English at Columbia University tells his own story.

"The Measurement and Appraisal of Adult Intelligence", by David Wechsler; Fourth Edition; 1958. Baltimore: The Williams and Wilkins Company. 9" × 6", pp. 314, with illustrations. Price: 55s.

The author is Chief Psychologist at the Bellevue Psychiatric Hospital, New York.

"Pathology for the Physician", by William Boyd, M.D., M.R.C.P., F.R.C.P., F.R.S., LL.D., D.Sc., M.D.; Sixth Edition; 1958. Philadelphia: Lea and Febiger. Sydney: Angus and Robertson, Limited. 10" × 6¾", pp. 900, with 149 illustrations and 12 plates in colour. Price: £9 12s. 6d.

This is a new book rather than a new edition, as a great deal of it has been rewritten.

"External Collimation Detection of Intracranial Neoplasia with Unstable Nuclides", by G. M. Shy, M.D., M.R.C.P., R. B. Bradley, B.S., and W. B. Matthews, jnr., B.S.; 1958. Edinburgh and London: E. and S. Livingstone, Limited. 9¾" × 6¾", pp. 152, with 15 illustrations. Price: 30s.

A highly specialized treatise.

"Medicine and the Navy, 1200-1900", by J. J. Keevil; Volume II (1649-1714); 1958. Edinburgh and London: E. and S. Livingstone, Limited. 8¾" × 6", pp. 332, with illustrations. Price: 40s.

The author has attempted to show how the medical service of the navy developed under the Commonwealth and Protectorate and the later Stuarts into a corporate organization designed to meet needs distinct from those served ashore.

"The Role of Science and Industry", by D. P. Mellor; 1958. Sydney: Angus and Robertson, Limited. Canberra: Australian War Memorial. 9¼" × 6", pp. 750, with illustrations. Price: 30s.

This book (a volume in "History of Australia in the War of 1939-1945") describes the part played by scientists, technologists and factory workers in equipping the Australian forces.

"Nutrition and the Papuan Child", by H. A. P. C. Oomen, D.Sc., M.B., and S. H. Malcolm; 1958. South Pacific Commission Technical Paper No. 118. Nouméa, New Caledonia: South Pacific Commission. 10" × 8", pp. 153, with 75 illustrations. Price: 8s. sterling.

The authors' idea was to start from the nutrition and diet of infants and their mothers, to study under the various ecological conditions the character and availability of the food resources of the population generally and, finally to discuss the over-all subsistence economy of the areas concerned.

The Medical Journal of Australia

SATURDAY, SEPTEMBER 27, 1958.

AUSTRALIA AND THE WORLD MEDICAL ASSOCIATION.

DR. L. R. MALLEN, of South Australia, was elected Chairman of the Council of the World Medical Association at the eleventh General Assembly of the Association held recently in Copenhagen. This is gratifying to the British Medical Association in Australia and, no doubt, to Dr. Mallen, who has fully earned the honour by his service on the Council of the World Medical Association since 1951; but, more than that, it is fitting, since the Council of the World Medical Association is to meet in Sydney in March, 1959.

The World Medical Association is becoming well known in Australia, particularly as a result of the visit in 1955 of the Secretary-General, Dr. Louis H. Bauer, to whom much of the credit must be given for the great progress the World Medical Association has made. During his visit Dr. Bauer delivered two major orations—the Henry Simpson Newland Oration,[1] on "World Medical Problems", at the ninth session of the Australasian Medical Congress (B.M.A.), and the Joseph Bancroft Oration,[2] on "The World Medical Association and its Relation to Medical Progress", at a meeting of the Queensland Branch of the B.M.A. Both of these are worth careful study by all who are interested in the medical problems of our modern world. They bring home particularly the constructive attitude towards these problems which is being taken by the World Medical Association, and the inescapable responsibility of national medical associations and of individual members of the medical profession towards their colleagues and the practice of medicine throughout the world. The World Medical Association, it may be pointed out, is a federation of the national medical associations of the world and is designed primarily for the interchange of information among them. Its objectives have been defined in the following terms:

> (i) To promote closer ties among the national medical organizations and among the doctors of all the world by personal contact and all other means available.
>
> (ii) To maintain the honour and protect the interests of the medical profession.
>
> (iii) To study and report on the professional problems which confront the medical profession in the different countries.
>
> (iv) To organize an exchange of information on matters of interest to the medical profession.
>
> (v) To establish relations with, and to present the views of the medical profession to, the World Health Organization, U.N.E.S.C.O., and other appropriate bodies.
>
> (vi) To assist all peoples of the world to attain the highest possible level of health.
>
> (vii) To promote world peace.

[1] M. J. AUSTRALIA, 1955, 2: 473 (September 24).
[2] M. J. AUSTRALIA, 1955, 2: 877 (November 26).

In a recent brief but comprehensive article on "International Organizations and the Practice of Medicine", Stanley S. B. Gilder[1] has pointed out that medical men have always shown themselves to be as gregarious as the members of any other profession, if not more so. As a result many groups have grown up, ranging from the medical society founded in an officers' prison camp in World War II, mentioned by Gilder, to the great national medical associations and now a surprising number of international organizations. Amongst the last-mentioned are two great groups—the government-inspired organizations and those formed voluntarily by the medical profession. There is no necessary conflict between the two groups. Certain activities are proper to the first group and probably can be carried out only by them, with their direct access to governments. On the whole, as Gilder comments, it may be said that in the past the effect of international health organizations on the practice of medicine has been a profoundly beneficial one in such fields as epidemiology, control of communicable disease, biological standardizations, unification of pharmacopœias and the collection of statistics. It is when these organizations encroach on the field of the practising doctor that difficulties may arise, just as they do when national governments seek to dominate the free practising profession. The experience of thoughtful doctors is that such a situation is no more in the interest of the patient than in that of the doctor. In the final picture the highest standards of medical practice are inevitably linked with the freedom of the practitioner. Thus it is no anomaly that the aims of the typical national medical association, such as the British Medical Association, are two, and face in opposite directions: the advancement of medical science and the maintenance of the harmony and interest of the medical profession. Rightly interpreted these are complementary and necessary to one another. As Gilder points out, the aims and activities of the World Medical Association fall within the scope of these two aims, and its supporters may therefore with every justification press forward and engage in their undertaking, in the words of Charles Hastings, founder of the British Medical Association, "with the zeal and alacrity of men anxious for the good of mankind".

The aims and ideals of the World Medical Association are such as to evoke a ready echo from the medical profession in Australia, and it is pleasing to record the active support, both moral and financial, which Australian doctors have given to the World Medical Association. Because of the distance involved it has not been practicable up till now to arrange a major World Medical Association function in Australia. A suggestion made a few years ago by the Federal Council of the British Medical Association in Australia that the General Assembly of the World Medical Association should be held in Australia was turned down, partly at least, for this reason. Even to bring the Council out here is quite a large undertaking, but it is now in sight. This distinguished group of our colleagues, some of them men whose names are household words in medicine, including the editors of the *British Medical Journal* and *The Journal of the American Medical Association*, may be confidently assured of a sincere welcome and warm hospitality. To the medical profession in Australia their coming will provide stimulus and encouragement.

[1] *World M. J.*, 1958, 5: 23 (January).

Current Comment.

IDIOPATHIC STEATORRHŒA AND THE GLUTEN-FREE DIET.

IN 1952 Charlotte M. Anderson and her colleagues at the University of Birmingham, following on reports from Holland by Dicke and by Weijers and van de Kamer, reported[1] rapid improvement in the clinical and biochemical state of children suffering from cœliac disease when they received a gluten-free diet. Deterioration followed reintroduction of gluten into the diet. The changes in the children investigated and treated were described as "very similar to those in adult idiopathic steatorrhœa".

Since then increasing attention has been paid to the disorder or group of disorders with which such terms as "cœliac disease", "idiopathic steatorrhœa" and "non-tropical sprue" are associated. This had led, amongst other things, to a critical examination of these terms, which were devised when much less was known about the matter than at present. Recently W. T. Cooke[2] has spoken strongly for the term "adult cœliac disease", pointing to the cogent clinical evidence that cœliac disease (of children) and idiopathic steatorrhœa are basically the same disorder; in particular he instances (i) the high incidence of previous cœliac disturbance in adult patients presenting with idiopathic steatorrhœa and (ii) the similar reactions to gluten-free diets. The second of these he regards as a therapeutic test, of value in differentiating adult cœliac disease or idiopathic steatorrhœa from other conditions which simulate it clinically, such as tropical sprue, regional ileitis and pancreatogenous diarrhœa.

The therapeutic value of a gluten-free diet for sufferers from idiopathic steatorrhœa is indicated in a study by J. M. French, C. F. Hawkins and Nadya Smith,[3] who treated 22 adult subjects. The treatment consisted in the administration of a diet free from the gluten of wheat and rye. Where necessary, to hasten recovery, treatment was also directed to correction of anæmia. Sixteen patients recovered; six failed to improve. After apparent recovery five patients were given gluten with the result that they quickly relapsed. In these cases, among other changes, the fæcal excretion of fat increased. Two healthy students were given 30 grammes of gluten powder a day without any effect on fat excretion. French and his colleagues note the difficulty of controlling the diet in some cases; for example, one patient secretly took "arrowroot" biscuits and induced a relapse, for in England "arrowroot" biscuits are made mainly from wheat flour. These investigators are unable to provide an explanation for the failure of the treatment in six cases. They mention the following possibilities: (i) the period of trial may not have been long enough; (ii) the patients may have been sensitive to substances other than gluten of wheat and rye; (iii) deficiency of vitamins may have prevented recovery; (iv) infection of the gut with pathogenic organisms may have occurred in the presence of large quantities of unabsorbed food; (v) some other disease may have been responsible for the non-absorption of fat. They reach three main conclusions: that in most cases the basic cause of idiopathic steatorrhœa is a reaction to wheat gluten; that a favourable response to dietary measures is slower in adults than in children, and the treatment cannot be regarded as failing unless it has been employed for at least six months; and that once "normal health" has been restored, the gluten-free diet must be continued indefinitely to avoid relapses.

Cooke suggests that it is justifiable to assume, should clinical cure result from institution of a gluten-free diet, that the patient is suffering from adult cœliac disease. However, he goes on to point out that the contrary assumption is not necessarily true. The effect of gluten appears to be quantitative, and even in patients in whom fat absorption has returned to normal, the result of the folic-acid-excretion test (which with intestinal biopsy he regards as diagnostically important) may remain abnormal. He comments: "The possibility therefore remains that there is a 'constitutional enzymatic defect' which is rendered evident by gluten administration, but which in some patients may be so gross that it cannot be corrected even by a gluten-free diet. From hæmatological effects and the persistently abnormal folic-acid excretions, gluten might be postulated as acting as a 'blocking' agent of an underlying metabolic disorder, possibly of the pteroyl-glutamate complex."

As a practical epilogue to this discussion, attention should be drawn to a detailed article by R. F. Fletcher and Marjorie T. McCririck[1] on how to prepare gluten-free diets that are pleasant and nutritionally adequate.

[1] *Lancet*, 1952, 1: 836 (April 26).
[2] *Brit. M. J.*, 1958, 2: 261 (August 2).
[3] *Quart. J. Med.*, 1957, 26: 481 (October).

PYGMY RACES.

ANY anthropological article under the name of R. Ruggles Gates, of Boston, Massachusetts, we expect to exhibit the full vigour of scientific research, and the reader of his latest pronouncement on African pygmies[2] will not be disappointed. Originally this investigation was directed towards a small colony in Western Uganda, but a wide extension in time and place developed, so that the origin and relationships of pygmies in general came under review. In Africa pygmies were once found over the whole continent; they are depicted in ancient Egyptian monuments, and early explorers have left accounts which could be applied only to true pygmies. Dwarfish stature can arise from malnutrition, but this is not transmitted genetically, whereas the true pygmy is to be regarded as resulting from a form of achondroplastic mutation and not as a pathological product. The pygmies with their mahogany skin and hairy bodies appear to be one of the oldest races in Africa, and were probably derived from a tall ancestral race. It is likely that the negro race originated later from the same stock by mutations which brought blackness of skin and reduced hairiness. The article gives a wealth of detail in anthropological measurements and is likely to remain authoritative for some time. The author refers to pygmies in New Guinea; and if his present visit to Australia gives rise to an investigation on our aboriginal population and the natives of New Guinea, we may confidently expect a contribution to physical anthropology of outstanding merit.

VIROLOGICAL DIAGNOSIS.

OF the many changes which have taken place in the past twenty-five years, the metamorphosis which has transformed the subject of virology is among the more spectacular. In this period it has grown from an infant appendage of bacteriology to full adult stature as an independent science. In a recent paper[3] F. S. Cheever reviews one aspect of this growth, namely, the changing status of virological diagnostic services.

Twenty-five years ago but one viral disease, rabies, was subject to routine laboratory diagnosis, by means of microscopic examination of infected tissues for the presence of the pathognomonic Negri bodies, first described in 1903. Today the majority of common viral infections may be diagnosed on a practical basis in any well-equipped virological laboratory, by demonstration of specific inclusion bodies, by isolation of the virus itself, or by demonstrating by serological methods the formation of antibodies against a specific virus. By the first method rabies, variola and vaccinia may be diagnosed. The demonstration of viral agents is carried out by injecting laboratory animals, for instance, the white mouse, in which animal many neurotropic viruses may proliferate in the central nervous system after intraspinal or intracerebral inoculation. Viscerotropic and respiratory viral agents may multiply in the white mouse after parenteral injection. Guinea-pigs,

[1] *Brit. M. J.*, 1958, 2: 299 (August 2).
[2] *Acta genet. med. et gemel*, 1958, 7: 159 (April).
[3] *J.A.M.A.*, 1957, 165: 2059 (December 21).

hamsters and rabbits are also used. For poliomyelitis, the monkey has been used, and for the Coxsackie group of viruses a suckling mouse. In these animals the presence of the virus has been shown by the development of a characteristic type of disease or recognition of specific pathological changes at autopsy. In the central nervous system relatively non-specific perivascular inflammatory lesions may aid diagnosis after the inoculation of animals with a variety of neurotropic viral agents. The use of the developing chick embryo for the growth of viral agents dates from 1931 and was popularized by Burnet and his collaborators in Australia. Evidence of infection of the embryo may be deduced from the death of the embryo with pathological findings suggesting a general type of viral infection. Pock viruses such as those of variola, vaccinia and herpes simplex produce a characteristic pock when inoculated on the chorio-allantoic membrane of the developing chick embryo. Viruses of influenza, mumps and pneumoencephalitis (Newcastle disease) produce hæmagglutinins to be identified by serological tests carried out with specific antiserums. With larger viruses of the psittacosis-lymphogranuloma group, direct microscopic examination of the infected yolk sac may identify the organism. This has led some purists to attempt to separate this group from the true viruses. Tissue cultures of human adult or embryonic cells have been used in propagating poliomyelitis virus. The antibiotics penicillin, streptomycin and nystatin have helped in the propagation of viruses, being used to destroy organisms which contaminate the inoculum. Even fæcal suspensions may be inoculated into embryonic eggs or tissue cultures, after preliminary exposure to small amounts of penicillin and streptomycin. Serological techniques, neutralization, complement fixation and, rarely, agglutination are also of value in identifying viral agents. A new hæmagglutination-inhibition test has proved useful in identifying the viruses causing mumps and influenza. The demonstration of the formation of antibodies against a specific virus during the course of an infection is now much used in diagnosis. The method is simplified by the fact that the viral antigen can be prepared ahead and stored in a frozen state. Quite a number of antigens and antisera are available commercially.

CARCINOMA OF THE APPENDIX.

PERUSAL of articles in American surgical journals at times gives the impression that American surgery tends to be radical. As an example of this may be cited a paper by J. Rabinovitch *et alii*,[1] in which the authors describe four cases of primary carcinoma of the appendix. Two of these were discovered incidentally in the course of operations on other viscera, one presented as a case of acute appendicitis, and in one the appendix was incarcerated in a strangulated femoral hernia. In each case the diagnosis was made on subsequent examination of the diseased organ, and each patient was advised to submit to a hemicolectomy. Three of the patients assented, were subjected to a second operation, and have remained well since; the fourth patient refused, and is alive and well after 12 years. In no instance was there evidence of spread beyond the appendix. These authors support their advocacy of routine hemicolectomy for all patients suffering from carcinoma of the appendix with the statement that "a general review of the literature shows rather poor results with local management of even the apparently less malignant lesions". However, their treatment of the subject appears somewhat superficial. They state that "tumours of the appendix may fall into two groups, namely, those which grow toward the lumen, and those which grow toward the mesentery". One of their tumours was diagnosed as a mucous-cell carcinoma, the other three are stated to have been adenocarcinomata. However, one is left wondering whether those authors appreciate the importance of an exact histological diagnosis, as it is difficult to relate their discussion to an authoritative review of the subject by W. B. Scimeca,[2] who states that there are three types of carcinoma of the appendix, namely, the carcinoid type (argentaffin carcinoma), the cystic or malignant mucocele type and the colonic type. The carcinoid type comprises 90% of malignant lesions of the appendix, and it is slow growing and rarely metastasizes. Scimeca states that almost all carcinoids can be cured by simple appendicectomy. The papillary mucous cyst adenocarcinoma causes simple obstruction, which results in distension of the appendix (malignant mucocele) which may reach a remarkable size. If it ruptures, peritoneal implantation of malignant mucus-producing cells results, causing the condition known as "jelly belly". However, this tumour does not metastasize by way of the blood and lymph streams; so Scimeca considers that if it is discovered before the mucocele ruptures, it is sufficient to remove the appendix and an adjacent portion of the caecum. The colonic type of carcinoma is the rarest of the three, and is the most serious. This tumour usually originates in a polypus; if it is still confined to the tip of this, appendicectomy is sufficient; but if ulceration and invasion have occurred, hemicolectomy is indicated.

Published reviews suggest that carcinomata of the appendix occur in between one in 500 and one in 200 of all appendices removed surgically. However, as already stated, the great majority of these are carcinoid tumours. Of the rarer tumours, about 30 cases of malignant mucocele have been seen at the Mayo Clinic since 1910 (benign mucocele of the appendix is found much more frequently), and only about six cases of carcinomata of the colonic type in which ulceration and invasion had occurred. Another very rare tumour of the appendix is primary lymphosarcoma; this has been seen twice at the Mayo Clinic.

[1] *Arch. Surg.*, 1957, 75: 122 (July).
[2] *Proc. Staff Meet., Mayo Clin.*, 1955, 30: 527.

ACOUSTIC NEURILEMMOMA.

IT seems a sad reflection on otology that a review of 300 cases of acoustic neurilemmoma by K. Cambon and F. R. Guilford[1] has revealed that only 10% were diagnosed by otologists. It is pointed out that these cases almost invariably present with symptoms caused by eighth nerve dysfunction. In such instances a strong suspicion of tumour is always wise. Hearing loss and tinnitus are more prominant than vertigo, which may be entirely absent. The hearing loss may be variable and may even show periods of return to near normal levels. Nystagmus may be very variable. These symptoms may precede the development of those suggesting intracranial disease by as much as 22 years, the average being one to three years. Findings additional to dysfunction of the eighth nerve in a series of 150 cases included corneal anæsthesia in 126; this was usually ipsilateral, but occasionally bilateral. Facial paræsthesiæ, headache and spontaneous nystagmus were frequent in occurrence. Facial weakness was observed in one-third of the series, but paralysis was complete in only one case. Visual disturbances occurred in 47 cases. A positive Rhomberg sign occurred in 106, and positive X-ray findings were noted in only 64 (42%). Almost invariably the caloric test revealed a non-functioning or hypoactive labyrinth. Other writers, however, have recorded 4% to 10% of cases in their series as giving normal caloric responses. As a general rule audiometric findings show a pure nerve loss, but no typical audiogram can be described. It is said that in the early stages low tone losses are common. The recruitment of loudness phenomenon is observed in end organ lesions, but it is likely to be absent in tumours of the cerebello-pontine angle. The tumour is almost invariably covered by an arachnoid cyst. This may, by the continued addition of fluid, reach such a size as to cause pressure effects of its own, and these effects may be variable. Apart from pressure effects upon the fifth and seventh nerves, the other cranial nerves are but rarely involved, and then only late in the course of the disease. Pontine and cerebellar compression may arise and superimpose confusing signs. Obstructive dilatation of the ventricles may lead to papilœdema and cerebral herniation.

[1] *Arch. Otolaryngol.*, March, 1958.

Abstracts from Medical Literature.

OBSTETRICS AND GYNÆCOLOGY.

Tuberculous Salpingitis.

M. HAINES (*Am. J. Obst. & Gynec.*, March, 1958), in the Joseph Price Oration, discusses tuberculous salpingitis. Tuberculosis of the female genital tract attacks the tubes in nearly every case, but the uterus in about only half. The tubes might be tuberculous in not less than 10% of cases of infertility. Of women found to have genital tuberculosis, 80% are between the ages of 20 and 40 years. Lesions of the tube may be classified as (i) normal in macroscopic appearance, (ii) exudative, (iii) adhesive, (iv) nodular. Pregnancy may occur in the tube either before the diagnosis is suspected or after treatment. Figures indicating the frequency of tubal pregnancy in tuberculous salpingitis vary from no case of tubal pregnancy in 200 cases of tuberculous salpingitis to three cases of tuberculous salpingitis in 302 tubal pregnancies. The differential diagnosis includes sarcoid, foreign body, leprosy, actinomycosis, bilharziasis, infestation with *Enterobius vermicularis*. The route of infection is generally hæmatogenous, and tubal function is generally impaired. Surgery today can be modified by the employment of specific drugs, which in some cases offer a hope of cure and in others make operation safer, but the advisability of performing plastic operations on tuberculous tubes has been questioned. Specific drug treatment must be maintained for a long time, pre-operatively for three to 12 months and for at least 12 months after operation. At all times it must be remembered that the patient has tuberculosis, rather than that she should be treated as merely suffering from infertility, a blocked tube, or a tubo-ovarian mass. Tuberculous salpingitis, like endometriosis, is hard to recognize, and an awareness of it is imperative, especially in cases of sterility. Endometrial curettings may help in the diagnosis, but many tubes which at operation appear normal or show mild inflammation turn out to be tuberculous if examined microscopically. Pregnancy, though attended by hazards, may be hoped for after therapy.

The Incompetent Cervix.

R. H. BARTER *et alii* (*Am. J. Obst. & Gynec.*, March, 1958) discuss the surgical treatment of late abortion or premature labour occurring after the sixteenth week of gestation as a result of cervical incompetence. The clinical picture is sudden loss of amniotic fluid between the sixteenth and twenty-eighth weeks of pregnancy, not preceded by painful contractions, and rarely occurring in a first gestation. These patients usually have had damage to the cervix from a dilatation and curettage, from cervical lacerations in a previous delivery, or as a result of surgical amputation done at the time of a previous gynæcological operation. The incompetent cervix becomes effaced and dilated between the fourteenth and twenty-sixth weeks of gestation. The process may be slow or rapid, but when it occurs the membranes can be seen bulging at the level of the external os or projecting into the vagina through the partially dilated cervix. In a completely typical case the fœtal membranes are under no apparent tension, the uterus is not irritable, and the dilatation appears to be a passive process. The authors state that the purse-string operation for the correction of an incompetent cervix seems to be the most physiological of the procedures described to date. The best results have been obtained when the operation is done between the fourteenth and eighteenth weeks, before the cervix has begun to dilate and before amnionitis has developed. A purse-string suture is inserted in the tissues of the cervix at the level of the internal os. The suture material used has been fascial graft, but the authors are now using a synthetic dacron mesh. The patient is kept in bed for 10 days, after which ambulation is gradually resumed. Most of the authors' patients have also been treated with large doses of progesterone. After full activity has been resumed, the cervix is examined under sterile conditions every two weeks. In successful cases Cæsarean section is performed at the thirty-eighth week. It is thought that by this procedure a single cervical operation may suffice for more than one gestation.

Hydatidiform Mole.

B. J. LOGAN AND L. MOTYLOFF (*Am. J. Obst. & Gynec.*, May, 1958) present a clinical and pathological study of 72 cases of hydatidiform mole, representing an incidence of one in 99 abortions or one in 974 pregnancies. In the early stages there were no clinical features that permitted a definite diagnosis. In only 22 cases was a mole suspected or diagnosed pre-operatively, positive diagnosis being made only after portions of the mole had been expelled or obtained by curettage. Spontaneous expulsion of the mole occurred in 12 instances. As soon as the diagnosis of mole was established treatment was directed to evacuation of the contents of the uterus. In the majority of cases abortion was imminent and occurred spontaneously. Pitocin was useful in effecting expulsion of the mole. A careful curettage was performed in all cases after spontaneous or induced abortion of the mole, except when hysterotomy or primary hysterectomy was performed. After removal of the mole, quantitative determinations of chorionic hormone were made at weekly intervals until the results were negative, and thereafter at monthly intervals for at least one year. Periodic X-ray examinations of the chest were also made. In most cases the chorionic hormone ceased to be demonstrable within two months. In eight cases evidence of malignancy was found, at intervals of one week to four months after detection of the mole. Final diagnoses in these eight cases were chorioadenoma destruens in five, choriocarcinoma in one, and metastatic moles in the lungs of two patients. Hysterectomy was performed in these eight cases, and there was no death or recurrence in the series. The authors were unable to find any correlation between the histological pattern of the mole and its potential malignant tendency.

PÆDIATRICS.

Distal Colonic Motility.

M. DAVIDSON AND C. H. BOWER (*Pediatrics*, May, 1958) describe three patients with fæcal impaction and soiling in whom a short area of achalasia proximal to the anus was demonstrated by motility studies. They point out that since Swenson described his operation of resection of the narrowed segment of bowel in Hirschsprung's disease variations in the clinical pattern of this disease have been reported. Thus cases of aganglianosis extending throughout the entire small and large intestines have been described; in others, variable lengths of intestine may be involved with skip areas between; cases have been reported in which very limited areas of aganglianosis extend from the internal anal sphincter to short distances within the lower rectum, and to this condition the name of megarectum has been given. The authors point out the importance of distinguishing between constipation due to organic achalasia and constipation due to simple functional impaction of fæces in the rectum. The latter is very much more common and results from perpetual neglect in the toddler or from psychological causes. It is quite the commonest reason for fæcal soiling in the infant and young child. In the three patients described in this report fæcal impaction in the rectum with soiling was present, but motility studies carried out by the passage of catheters into the bowel and the estimation of pressures after the administration of "Mecholyl" indicated that there was a short area of achalasia proximal to the anus. Surgical resection of this achalasic segment resulted in improvement in all three children, although ganglia could be demonstrated in this resected segment in each child. These ganglia were less numerous than in normal bowel.

Moniliasis in Pædiatrics.

B. DAVIES (*Am. J. Dis. Child.*, September, 1957) reviews the ways in which monilial infections may present in childhood and infancy. The infection may be caused by any species of *Candida*, but it is usually caused by *C. albicans*. The disease may range from transient benign stomatitis, comprising the majority, to a generalized fatal disease. Dissemination occurs by contiguity, and by way of the blood stream and lymphatics. Skin lesions in infants are more superficial and usually cover larger areas than those in older children. Moniliasis seems to be the cause of perianal dermatitis in new-born infants more frequently than is generally realized. Surface spread from the mouth may result in the formation of pseudomembranes in the œsophagus, ulcers and perforations of the intestines, or pulmonary lesions. Severe vomiting, diarrhœa, a toxic appearance, shock with low temperature and enlargement of liver and spleen may suggest systemic moniliasis due to blood stream spread. This form of the disease usually runs an acute and frequently fatal course in infants. A history of symptoms of superficial involvement of the mucous membranes and skin may often help to suggest the diagnosis. Abscesses in

practically any part of the body may be found on autopsy. *Candida* infections beyond infancy usually have a more chronic course and often represent a continuation of infantile infection. The commonest sites of skin infection are the face and the extremities. Cutaneous granulomata, abscesses and localized granulomatous and necrotic organ lesions as well as meningitis, may develop in cases of long standing. Allergic reactions may result. *Candida albicans* is often found in the mouth and fæces of infants, sometimes without causing clinical symptoms. It is almost never present on normal skins or in skin disorders of other ætiology. Familial vaginitis in the mother, contaminated hands and objects, and air-borne infection are some of the sources of moniliasis in infants. A familial incidence of the infection has been noted. The author discusses factors predisposing to thrush, and the various forms of treatment available. He regards nystatin as the best of these. In infants and children oral administration of this antibiotic should always accompany topical applications, since many patients have foci of infection in the gastro-intestinal tract. Treatment of pregnant women with monilial vaginitis might prevent thrush in the new-born. Isolation of infants with thrush is advised.

Electroencephalography in Subdural Hæmatoma in Infants.

M. STREIFLER, E. FRUENDLICH AND A. J. BELLER (*Am. J. Dis. Child.*, January, 1958) describe the electroencephalographic findings in 14 surgically verified cases of subdural hæmatoma and effusion in infants. Abnormal curves were obtained in 13 cases. These abnormalities consisted in a reduction of voltage ("flattening") and slowing of the electrical brain activity in 11 cases, while there was a general diffuse slowing in only two cases. In one case the electroencephalogram was rated as within normal limits. The high incidence of abnormal records in the series indicates the value of the electroencephalogram in the diagnosis of this condition. Although the electroencephalogram changes are not specific or pathognomonic for this clinical entity, this harmless and relatively simple examination may help in the diagnosis of subdural hæmatoma and effusion in infants. In suspected cases, an abnormal electroencephalogram record makes exploration by subdural puncture or burr holes mandatory; a normal electroencephalogram justifies a more conservative approach.

Babinski and Plantar Grasp Reflexes in Infancy.

H. F. DIETRICH (*Am. J. Dis. Child.*, September, 1957) reports a study of the Babinski and plantar grasp reflexes in a group of 103 infants examined repeatedly from birth to one or two years of age. He reports that a spontaneously appearing Babinski's sign, which is seen principally in the new-born period, is not a reflection of any clinically recognizable pathology. During the first six months of life Babinski's sign can often be elicited by stroking the outer side of the sole, but the sign appears and disappears, sometimes repeatedly in the same infant, and the author believes that lack of myelinization of the nervous system is not a rational explanation for its presence in the young infant in view of this irregularity of its appearance in any individual. After six months of age the sign is relatively uncommon. The Babinski sign must not be confused with the plantar grasp reflex, consisting of a forceful flexion of all the toes, and elicited by stimulating the ball of the foot. The plantar grasp reflex is remarkably constant and active during early infancy and usually disappears between six and 12 months of age.

Intrathoracic Tension in Infancy.

J. GILBERT AND R. MYERS (*Arch. Surg.*, March, 1958) discuss five cases of children, of ages ranging from six days to four months, all *in extremis* due to progressive tension states from lung cysts. These patients had respiratory difficulty and cyanosis requiring continuous oxygen and all required urgent thoracotomy with removal of the affected lobe of the lung. Recovery was immediate and the post-operative courses essentially uneventful. Subsequent examinations showed that the remaining lung tissue had expanded to fill the pleural cavity. In four instances the cystic process was found to be limited to a single lobe. In one it was localized to the upper portion of the lung. Three of the resected specimens contained typical bronchogenic cysts lined by the columnar type bronchial epithelium. One lobe was found to consist entirely of multiloculated cysts lined with alveolar epithelium.

ORTHOPÆDICS.

Fractures of the Tibial Shaft.

H. ELLIS (*J. Bone & Joint Surg.*, February, 1958) states that there have been very few controlled studies of the rate of fracture healing. His present study is to investigate the effect of severity of injury, skeletal traction, and distraction on fracture healing. This investigation covered a consecutive series of 576 tibial shaft fractures seen at the Royal Infirmary, Sheffield. Children beneath the age of 16 years were excluded, as they all healed rapidly, and the adult group of 343 was investigated. The most important factors were those which emphasized the severity of the original injury, such as severe open wounding, gross comminution and complete displacement of the fracture. He also found that distraction, that is separation of the fracture end by a gap of one-sixteenth of an inch or more, was another factor in the delay of healing. Five weeks extra were required on an average. Traction alone produced no significant difference, nor did the presence of infection, which occurred in only five of the open wounds. He found that the site of the fracture was also of little significance. In fractures below the tibial condyles union occurred in every case. In the remaining part of the shaft the site had no effect in itself.

Tuberculosis of the Spine.

R. ROAF (*J. Bone & Joint Surg.*, February, 1958), in his editorial, states that most orthopædic surgeons in England have been brought up to believe that it was wicked to perform planned operative exposure of uncontaminated tuberculous foci. He points out that in the last decade many surgeons have operated on para-vertebral abscesses and tuberculous foci in the spine. From these surgeons we have learnt four things: (*a*) that there was dramatic relief of pain and improvement of the patient's general condition; (*b*) that in the abscess much material was found that would be considered a bar to healing in non-specific osteomyelitis; (*c*) that living organisms are present, hidden in avascular areas impervious to systemic chemotherapy; (*d*) that X-ray diagnosis is deceptive, and that lesions that appear healed are in reality often active. The author reminds the reader that there are still many unknowns. When should the surgeon intervene? How long should conservative treatment be persisted in before there is surgery? He also remarks that there is considerable difference of opinion as to the various techniques of access, particularly to the cervical and upper thoracic regions. When the surgeon has opened, there is again a variety of opinions as to what should be done. Some only drain. Others completely excise the abscess and its wall. Others carefully cleanse out the bone cavity. Others go further and fill the cavity with bone chips. There is a difference of opinion as to the value of local instillation of streptomycin. Some advocate it, others abhor it. He points out that there is also a number of people who consider that tuberculosis is being eliminated and that it really does not present a great problem at present, particularly where a welfare state cares for the patient and supplies antibiotics without cost. However, this is not so in primitive countries, and it is important to know the best and most economical method of handling the condition. The author also points out that bone tuberculosis is a local manifestation of a systemic disease.

Excision of the Patella.

L. H. DUTHIE AND J. R. HUTCHINSON (*J. Bone & Joint Surg.*, February, 1958) state that there is clearly a difference of opinion as to the after results of total and partial excision of the patella. Hence they reviewed the results of these operations performed in the Western Infirmary, Glasgow, over the last 10 years. There were 146 cases, and 72 of these were assessed by interview and X-ray examination. In this assessment the quadriceps development and power were graded and compared with the complaints and symptoms. There were three operations: excision of the distal pole, patellectomy after fracture, and patellectomy after chondromalacia. In the final assessment the best results came from total excision of the patella for chondromalacia. It was thought that this was because these patients were mostly in the younger age group. Total excision gave better results than partial excision; and it appeared that suturing the ligamentum patellæ to the anterior lip of the upper fragment tended to tilt the fragment and abrade the condyle, and so tend to the production of post-traumatic arthritis. The other finding was that the poorer results occurred more often when there was an associated calcification after the removal of the patella.

British Medical Association.

NEW SOUTH WALES BRANCH.

INTERIM REPORT ON MEDICAL EDUCATION: PART I.

THE following are the minority reports submitted by Professor Keith Inglis and Dr. George V. Hall as members of the Committee on Medical Education of the New South Wales Branch of the British Medical Association.

Minority Report by Keith Inglis, M.D.

I submit herewith a minority report by me in my capacity as a member of the Committee on Medical Education.

In expressing the following opinions I am in general agreement with the Council:

1. The establishment of a second medical school in New South Wales is necessary.

2. The second medical school should be an integral part of a university and should be connected with a university hospital on the same campus.

3. Limitation in numbers of medical students in medical schools is necessary for the maintenance of standards.

4. Adoption of different standards in medical schools should be avoided.

I differ from the Council, however, in regard to the site of the second medical school and the university to which it should belong. The Murray Committee is probably the most detached committee from which we are likely to have a pronouncement on this subject. The Murray Committee stated (p. 87, sect. 317, and p. 88, sect. 318): "There appears to be every justification for the establishment of a second medical school in New South Wales. . . . If there should be a new medical school, there is much to be said for placing this in the N.S.W. University of Technology"; and in section 318: ". . . it must be expected that the N.S.W. University of Technology will assume many of the features of a traditional university".

In my opinion universities should have a broad base, and the establishment of a Faculty of Arts and a Faculty of Medicine in the N.S.W. University of Technology, later to be named the University of New South Wales, would help that university to become a general university. It would also lighten the load on the University of Sydney.

The N.S.W. University of Technology was established some ten years ago, and no other university in New South Wales is at present in a position to help in solving the problem now under consideration.

For the above reasons, in my view, the second medical school should be established in the N.S.W. University of Technology, provided that university becomes a general university with a Faculty of Arts as well as a Faculty of Medicine, and its name is changed to University of New South Wales.

(Signed) KEITH INGLIS.

September 4, 1958.

Minority Report by George V. Hall, M.R.C.P. (Lond.), M.R.A.C.P.

In my capacity as St. Vincent's Hospital representative on the Committee on Medical Education, I submit herewith a minority report.

I am in general agreement with the B.M.A. Council on the following points:

1. I agree that the establishment of a second medical school in New South Wales is necessary. Having studied the facts and figures of the matter, I believe that this is an urgent necessity if a good standard of medical education is to be maintained.

2. I agree that a medical school should be an integral part of a university. Medicine is a branch of higher learning, and as such should have this university association.

3. I agree that limitation in numbers of medical students admitted to medical schools is necessary for the maintenance of good standards in the future. However, the method of implementing this control should be very carefully considered. A special investigation of the method should be undertaken before this control is implemented, and adequate notice should be given that a quota system is to be introduced.

4. I agree that in the event of further medical schools being established, the adoption of different standards must be avoided and that curriculums should be coordinated.

I disagree with the B.M.A. Council's recommendations in the following respects:

1. I consider that the suggestion by the Government that the second medical school be established at Kensington on the campus of the University of New South Wales is a reasonable one. In doing this I accept the Government's assurance that a Faculty of Arts will be established at the University of New South Wales and that this university will assume the features of a traditional university.

2. I disagree that it would not be in the best interests of the State to have a medical school associated with a university where technologies are taught. It is to be remembered that at the University of New South Wales these technologies are taught by professors who are university graduates of high academic standing and that the undergraduates have matriculated at a standard as high, if not higher, than undergraduates at the University of Sydney. In this modern age there should be a growing appreciation of the importance and increasing status of these arts. It is difficult to see why these faculties should be debarred from associating with the so-called higher branches of learning, such as medicine and arts. In my opinion such an association could achieve nothing but benefit to all concerned.

3. I disagree with the proposed site of a new medical school: (*a*) in the area of Parramatta; (*b*) in the area in the Lane Cove Valley. Whilst this may theoretically be a sound plan for the future, it is, in my opinion, an unrealistic one for the urgent needs of the immediate present.

The Government is being asked to establish a university college with other faculties in a certain area, so that a medical school may be established at that site before it has been established that a university college is in fact an urgent necessity in that area. Surely it is more realistic to establish a medical school at an established university, where the land and administrative facilities are already available, and where there are already large teaching hospitals established and about to be established in the immediate vicinity.

A decision for the Kensington site must see the establishment of the second medical school more expeditiously and economically accomplished.

The B.M.A. Council's proposals for a university college and medical school at the sites they nominated may well be an excellent blue-print for the future. In ten or fifteen years' time this project may well be undertaken.

(Signed) G. V. HALL.

Congresses.

INTERNATIONAL DIABETES ASSOCIATION.

THE third International Congress of the International Diabetes Association was held at Dusseldorf, West Germany, from July 21 to 25, 1958. The following account of it has been supplied by Dr. J. Kempson Maddox.

This congress is organized by the Council of the International Diabetes Federation, as were its predecessors at Leyden and Cambridge six and three years earlier respectively, and has doubled in size at each session. The "I.D.F.", as it is called, is a union of all national diabetic associations or societies of the world and up to now has had its headquarters in London, under the presidency of Dr. R. D. Lawrence. Unfortunately, Dr. Lawrence has retired owing to ill health, and Professor Hoet, of Louvain, Belgium, has been elected President in his place. The Society has both professional and lay members, and the programme of each congress is similarly bilateral.

About one thousand participants attended this congress, which was held in the Ehrenhof, a series of handsome new buildings beside the Rhine, especially designed for fairs and exhibitions, which are a feature of this busy, progressive and centrally situated city. The organization of the congress had been done with characteristic German thoroughness, and many attractive ambulatory interpreters helped to overcome language difficulties, in conjunction with an efficient simultaneous translation service at each meeting. The Australian delegates were Miss Ruby Board, of Sydney, President of the Diabetic Association of Australia, and Dr. J. Kempson Maddox, of Sydney. Dr. Kate Mackay, of Melbourne, also attended the congress. The majority of the papers were given in German, but summaries in English had been prepared in advance.

The lay and scientific sections met separately for the most part, but combined on such topics as insurance problems,

drivers' licences, employment for diabetics and the suppression of quackery. Altogether 212 papers were read in three simultaneous sessions, 60 of which were concerned in some way with oral drugs in use or under investigation for the treatment of diabetes mellitus.

Mode of Action of Insulin.

The question of the mode of action of insulin was chiefly discussed by Dr. R. Levine (Chicago) and Dr. C. R. Park (Nashville, U.S.A.). Insulin appears to be able to act on the transport system present in all cell membranes, so as to effect rapid entry of glucose and structurally related sugars into the intracellular space. This also holds for galactose, dixylose and l-arabinose. Muscular work activates what is probably the same transport system. Park studied this mechanism on erythrocytes and isolated muscle of the rat heart. Hexoses and pentose compete for this mechanism, which is independent of glucose phosphorylation, and which places a limit for glucose utilization in both the normal and diabetic organism. Insulin accelerates the process in both directions. Hypophysectomy results in accelerated transport, while alloxan diabetes leads to inhibition of transport, presumably due to unopposed pituitary and adrenal factors, including somatotrophin.

Recent Research in Carbohydrate Metabolism.

Dr. de Duve and his associates at Louvain discussed the difficult question of the effect of insulin on the liver. They demonstrated an anabolic action of insulin on isolated liver slices from the normal animal. This effect was strongly inhibited in the diabetic animal, and was not immediately correctable by administration of insulin, although in the intact animal the liver responds as rapidly as the muscles to insulin deprivation. However, Ashmore *et alii*, by directly measuring glucose output from the liver, have recently shown that insulin is without direct effect upon hepatic glucose production. Such a process may be indirect and secondary to hypoglycæmia.

Permeases specific for various sugars exist in bacterial cell walls, according to a paper by Dr. B. L. Horecker, and thereby organisms concentrate sugar to a degree of as much as 2000 times the concentration in the surrounding substrate.

The influence of corticosteroids on carbohydrate metabolism was discussed by Dr. C. N. H. Long (Yale). It has generally been assumed that in starvation the role of the glycogenic adrenal steroids has been to accelerate the release of tissue proteins and, in consequence, of endogenous carbohydrate, but evidence exists that this may not be the primary action of these hormones. Increase of liver glycogen and blood glucose content may occur in the absence of any changes in protein metabolism, and are not dependent on the presence or absence of insulin. In the absence of the liver, either deprivation or excess of these hormones has very little effect on either protein or carbohydrate metabolism. Steroids probably have much more effect on hepatic as compared to peripheral carbohydrate metabolism.

P. J. Randle and G. H. Smith have studied the mechanism of insulin action in muscle by means of the isolated rat diaphragm. They have shown that anoxia or substances such as sodium salicylate which inhibit oxidative phosphorylation can, like insulin, increase the utilization of glucose by isolated diaphragm. This may account for the well-known anti-diabetic effect of salicylates.

Dr. D. B. Martin (Boston) has had the lipogenic effect of insulin under study. He has found that the effects on fat depots are immediate and extremely dynamic, oxidation to carbon dioxide and water or incorporation of glucose with the lipid occurring within fifteen minutes after giving insulin. There was no effect on the acetate substrate. While this is probably under hormonal control, there was no direct adrenal effect demonstrable. There are apparently two alternative pathways in the fat synthesis, and differences may exist biochemically in the deposition of liver and of peripheral fat.

In the discussion which followed, Dr. Levine described the function of the liver as regulatory rather than participatory. A glucose load of more than slight degree was probably not wholly worked over by the liver, in which glucose-6-kinase was present in large quantity.

Dr. de Duve disagreed and thought that the transport system might be "fragile" and that the size of the liver cells might be important.

Diabetic Angiopathy and Nephropathy.

The morphology of extrarenal vessels was taken up by Dr. Randerath (Heidelberg), who has investigated the limb arteries of diabetics by means of a variety of new stains and the use of polarized light. His contention is that an accumulation of high polymer mucosaccharides occurs in the external muscular coat of the diabetic vessel. The atheroid plaque in the diabetic subject contains more cholesterol ester and phospholipid, but less neutral fat, than that of the non-diabetic individual.

Dr. Kimmelstiel, whose name has been given to the well-know nephropathy of late diabetes, defended the specificity of this lesion, at least in its early phase. He drew attention to the thickening and exudate in the parietal layer of Bowman's capsule and in the basement membrane of the proximal convoluted tubule displacing the epithelium centrally. He advocated the use of modified aniline blue and PAS stains in order to demonstrate these changes best and to distinguish them from the non-specific "exudative" or embolic glomerular lesions found in other types of chronic renal disease. As a result of histochemical studies and electron microscopy, he believes that intercapillary glomerulosclerosis follows reabsorption of polymucosaccharides, lipoids and proteins, which circulate in the systemic blood-stream and settle out in the kidney and retina. He agrees that the clinico-pathological correlation is often difficult.

Dr. Adlersburg (New York) inclines to the view that both diabetes and the vascular complications of the disease are due to separate metabolic errors genetically determined. There appears to be no qualitative, but striking quantitative, differences between diabetics and non-diabetics in relation to atherosclerosis. In discussing the disturbed lipoid metabolism of even mild, seemingly well-controlled diabetics, Dr. Adlersburg referred to a syndrome of idiopathic hyperlipæmia, mild diabetes and steadily advancing vascular damage, often associated with xanthoma and relapsing pancreatitis. He was impressed with the importance of elevated serum triglycerides as an unfavourable finding in the diabetic, however mild, rather than observation of serum cholesterol or phospholipid.

Dr. P. A. Bastenie (Brussells) commented on the extreme rarity of vascular lesions and retinopathy in hæmochromatosis as compared with classical diabetes of the same severity and duration. He thought that vascular degeneration might even begin in the prediabetic phase. Clinico-pathological correlations revealed that the nephrotic picture was rare in the Kimmelstiel syndrome, and that in older subjects the clinical picture was usually that of a non-specific glomerulonephritis. Dr. Bastenie has also investigated the pattern of excretion of the 17-ketosteroids, but has not found any difference in diabetic women with and without vascular complications. However, he is convinced that the adrenal plays some part in the disturbed lipoid metabolism which precedes the clinical appearance of vascular disease.

Other speakers told of the results of chemical examination of the aorta in diabetics, which revealed much higher fat and lipid values than in controls. The same findings applied to veins.

Dr. Gerritzen held that vascular complications were less common in insulin-deficient diabetics. Of a group of diabetic children studied in New York, 56% showed elevated serum lipoprotein values, which precede vascular complications by a long period. Treatment with insulin and a high carbohydrate diet appears to delay their appearance.

Dr. Hardwicke (London) said that he had failed to find any normal foot flows in diabetics of more than two years' duration when studied by foot plethysmography after temporary vascular occlusion.

Dr. Ricketts (Chicago) described experiments in dogs which suggested that in younger animals arteriosclerosis is more common in poorly controlled diabetes.

Three new long-term diabetic anomalies were described by Dr. Lundbaek (Aarhus)—namely, osteoporosis, a scleroderma-like appearance of the palms and "areolar" exudates of the retina. Proliferative retinitis carried a bad prognosis, but was often slow in its progression.

Dr. H. F. Root (Boston) reviewed the problem of diabetic nephropathy. He stated that intercapillary glomerulosclerosis rarely if ever occurred in an isolated lesion. It included both degenerative and infectious elements. In young persons the nephrotic phase preceded that of azotæmia by about five years. An important diagnostic aid was the finding in the blood of birefringent lipid material in the form of small Maltese crosses under polarized light. He was convinced that this lesion, which is responsible for 60% of deaths in diabetes beginning in childhood, could be prevented or delayed by early persistent and continuous supervision.

Dr. Theil, an oculist from Frankfurt, showed a series of beautiful retinal photographs. He indicated that an early sign of diabetic retinopathy was the thickening and varicosity of the retinal veins.

The lessened frequency of diabetic gangrene requiring amputation was referred to by Dr. Mörl (Halle), and other speakers agreed that a conservative approach was now justified.

Insulin Assay and Insulin Resistance.

Dr. Taylor and Dr. Randle, using the rat diaphragm uptake method to determine the insulin content of blood plasma, have found that plasma may contain other substances which affect insulin uptake. Zone electrophoresis on columns of treated cellulose has been studied as a possible method of separating insulin. Insulin has been found to move with part of the albumin fraction, and this fraction has been found to stimulate the uptake of insulin by rat diaphragm.

Dr. Vallance Owen (London) described experiments which show that uncontrolled insulin-requiring diabetics have an inhibitor in their plasma for insulin which disappears after insulin treatment. The antagonism appears to depend on the presence of pituitary products and adrenal oxysteroids.

Dr. D. B. Martin and associates, using the epididymal fat pad of rats, have found that insulin added *in vitro* causes a fourfold increase in glucose uptake by this tissue; this can be used as a method of insulin assay for amounts as small as 10 microunits. The procedure can be performed simultaneously with several pieces of tissue from the same animal. Plasma insulin-like activity in normal human subjects in the post-absorptive stage ranges from 50 to 75 microunits per millilitre.

Dr. R. H. Williams described several insulin antagonists: first, the factor present in the alpha-globulin fraction of plasma in diabetic acidosis; second, the antibody which is developed by diabetics and non-diabetics after several weeks of insulin injections, and which migrates to the inter-gamma-beta globulin zone; third, antibody to insulin appearing in the alloxanized rat, which accumulates in the lipoid fraction, and for the persistence of which both growth hormone and hydrocortisone are necessary. The plasma of some diabetics exhibits insulin antagonism when they are not under good control, but none when they are stabilized.

Dr. Moinat has been able to quantitate insulin antibodies by a specific and sensitive hæmagglutination method.

A group of German workers have treated clinical insulin resistance by the daily administration of insulin by the intravenous instead of the subcutaneous route, and they claim a rapid lowering of insulin requirements.

Dr. W. Oakley (London) described two patients with marked insulin resistance whom he had treated with prednisone with dramatic improvement. He considered that this was due to a suppression of glucocorticoid production.

Dr. Buding (Berlin) has had success with a procedure resembling auto-hæmotherapy.

Oral Treatment of Diabetes.

Dr. A. Marble (Boston) recounted his clinical experiences with the use of tolbutamide. Selection was made on the basis of a four-hour single-dose sulphonyluria response test. The main factors concerned were the age of onset of the diabetes and the daily insulin requirement, which should not exceed forty and preferably twenty units. He advocated first an adequate trial of diabetic therapy alone, extreme care in the transfer from insulin to tolbutamide, in case the patient was not responsive to the latter, insistence on normal blood sugar levels, careful observation of the patient, and trial without tolbutamide if a stage was reached at which the dose was so low that the patient could probably be maintained on diet alone. The incidence of secondary failures was 6%, with untoward reactions in 1%. No changes were observed in liver function, and no changes attributable to tolbutamide were observed in the liver or pancreas *post mortem*.

One remarkable aspect of this discussion was the favourable experience of Continental observers in regard to the paucity of untoward effects following the use of carbutamide (BZ55).

Dr. Boulin and Dr. Rampert (Paris) have treated a series of 200 patients with this drug in doses of only 0·25 to 0·5 gramme, sometimes at intervals of two or three days, and stated that these doses seemed to be equally as efficacious as higher ones. They eliminated patients with hepatic or renal failure and selected patients who were stout and who had mild diabetes without ketosis. Results were excellent in 99 patients, good in 33, mediocre in 33 and poor in 35. No serious sequelæ were seen, but intolerance, particularly in the form of skin rashes, occurred in 26%. Leucopenia was seen in 30 patients, mainly during the first three months of treatment. Vitamin B appeared to reduce the incidence of side effects.

A mass survey by Dr. C. J. O'Donovan of several thousand patients in the United States, treated for up to two years with tolbutamide, revealed a remarkable absence of toxicity.

Dr. Russell Fraser (Hammersmith Hospital) said that a clinical trial revealed the responders to tolbutamide in twelve weeks, and that tests showed that those responding had near normal insulin values and normal peripheral utilization.

There was general agreement that tolbutamide and carbutamide act by direct stimulation of the islets of Langerhans. Tolbutamide itself has no direct effect on peripheral utilization, as shown by injection into the brachial artery, according to Dr. M. Miller (Cleveland). The uptake of glucose by circulating leucocytes is greater during tolbutamide administration.

Dr. A. and Dr. O. Sirek (Banting and Best Institute, Toronto) gave tolbutamide in doses of 30 to 100 milligrammes per kilogram to dogs for periods up to fourteen months. They observed an elevation in serum phosphatase concentration, which was followed by a fall in serum albumin and a rise in serum transaminase levels. Later some animals showed a fall in prothrombin level and became jaundiced. Histological examination revealed changes in the Kupffer cells.

Dr. I. Murray and Dr. I. Wang (Glasgow) advocated treatment by intermittent courses of sulphonyluria. Little or no evidence of toxicity was observed in patients treated in this manner by carbutamide.

Several new hypoglycæmic substances for oral administration were described by German speakers. One was "P607", a powerful agent which is used in doses only a quarter of those required for tolbutamide, and is free from the sulphonamide group, but is associated with gastric side effects.

Much interest was shown in phenethyldiguanide ("D.B.I."). According to Dr. O. J. Rafaelson, this substance caused a much higher uptake of glucose by the rat diaphragm than did carbutamide. Dr. Pomeranze and Dr. Gadlek (New York) reported their experiences with a series of 210 patients of all ages. Of those over 40 years of age, 70% were successfully treated, but in the younger group no patient was able to cease insulin entirely. Dr. Priscilla White (Boston) has also been impressed with results in children using D.B.I. in doses of from 10 to 200 milligrammes daily (0·5 to 1·0 milligramme per pound of body weight). She referred to the infrequency of hypoglycæmic shock and the apparent absence of toxic effects in her experience. Other speakers were less happy about the drug, which apparently abolishes aerobic glycolysis by interference with the citric acid cycle. Dr. J. Walker (Leicester) had treated 34 diabetics with P607 (chlorpropamide), with excellent control in 20, but gastric symptoms and leucopenia occurred in more than half his cases. Dr. A. Loubatières, a pioneer experimenter with the arylsulphonamides since 1942, remains convinced that these substances potentiate injected or endogenous insulin, that they cause immediate degranulation of the beta cells of the pancreas, and that they even ultimately stimulate the formation of new beta cells. Neither the liver nor any endocrine gland other than the islets is necessary for the hypoglycæmic effect of these drugs. He believes that if they are used in man before total diabetes occurs, occasional "cures" may result.

Diabetes in Pregnancy.

The discussion on diabetes in pregnancy was opened by Dr. J. P. Hoet (Louvain), who described changes in pattern in the glucose tolerance of the pre-diabetic pregnant woman, but these were of minor degree.

Dr. W. P. U. Jackson, of Cape Town, had noticed a slight elevation of the two to two and a half hour blood sugar reading. Two doses of cortisone given just prior to the test may reveal the abnormality, but cannot be relied upon. He considers that the best evidence of pre-diabetes is to be found in the pancreas of the infant, where the islets become "continents".

Dr. Hoet, junior, thought that vitamin A deficiency and thyroid depression were features of these women. Of a series of infants weighing over 10 pounds at birth, 31 had pre-diabetic mothers, 14 had pre-diabetic fathers and three had normal parents.

According to Dr. Reis (Chicago), uncontrolled diabetes leads to loss of fertility. The abortion rate is increased, and, according to Dr. P. White, only one pregnancy out of three is successful. She said that the pre-viable loss was 22%, eclampsia occurred in 33%, and some degree of hydramnios occurred in all. If ketosis was allowed to develop in the second or third month, all fœtuses died.

Dr. Solth, in a review of 380 cases, showed that the abortion rate rose in relation to the earlier onset of maternal diabetes.

Dr. Peel (King's College Hospital) referred to the evidence of placental insufficiency, as indicated by low levels of oxygen saturation in the fœtus and raised hæmoglobin levels, as well as a slow rate of removal of radioactive sodium injected into the uterine wall. This was most noticeable in the presence of hydramnios. He believed that rest for the mother was the greatest single restorative agent.

Dr. Reis stated that he had found no specific abnormalities in the histology of the diabetic placenta.

Dr. Oakley drew attention to the abnormalities in body weight and body length in infants dying within forty-eight hours of birth, as compared with infants born before the twenty-eighth week, from which he concluded that the factors responsible for this change must operate after this date.

By measurement of total body water and extracellular water, Dr. Osler (Copenhagen) was able to say that this was actually less in diabetic infants, and that their excess weight was due not to œdema but to fat.

Dr. J. M. Malins (Birmingham), in an interesting paper, remarked that the fœtal loss fell from about 30% to about 25% when one physician and one obstetrician joined in an agreed policy. In his hospital the fœtal loss under varied supervision was 36·4% and under unified control was 17·5%; in cases seen before the fifteenth week, the figures were 33·3% and 4·3% respectively.

This emphasizes the importance of good diabetic control from the day of conception. The rate of fœtal loss at the Royal Prince Alfred Hospital, Sydney, as computed by Dr. Parkin, was presented as 23% in a series of 112 pregnancies and were surpassed only by those of Dr. White and Dr. Malins.

Dr. Pederson (Copenhagen) stated that the greatest vulnerability to hypoglycæmic shock was found in the last trimester, and that the risk of fœtal injury was the greatest at this time.

Dr. Paley stated that with increased fœtal survival the problem of congenital defects became enhanced. He said that one in six offspring of diabetic mothers had some form of congenital defect, and that this had no relation to the family history, toxæmia or the severity of the maternal diabetes.

At the conclusion of this panel discussion Dr. P. White gave her latest statistics, covering 1700 pregnancies, 1060 of which had reached the twenty-eighth week, and in 900 of which the mother had been given sex hormones. The overall fœtal loss was 13%.

Miscellaneous.

Other papers dealt with enzyme chemistry, the chemistry of the oral anti-diabetic substances, psychiatric aspects, particularly of childhood, histo-chemical studies of the islets, the action of salicylates etc.

The lay sessions dealt with summer camps for children, insurance problems, unemployment, drivers' licences, schooling, apprenticeship etc., and will be described in due course in *The Australian Diabetic Journal*.

The social programme was generous and enjoyable, and all who attended agreed that the German Diabetic Association, under the presidency of Professor Oberdisse, had done a splendid job.

The next congress is scheduled for Italy in 1961.

Out of the Past.

In this column will be published from time to time extracts, taken from medical journals, newspapers, official and historical records, diaries and so on, dealing with events connected with the early medical history of Australia.

THE MEDICAL UNDERGRADUATE.

[From the *Australasian Medical Gazette*.]

FROM the presidential address to the N.S.W. Branch of the B.M.A. by Robert Scot Skirving, M.D., March 4, 1892.

It is with pardonable pride that I refer to the Medical School of our University with its well-equipped hospitals. We have a five years curriculum, and our students are mentally every bit as good, but not better, than the medical youth of European schools. Our purely scientific subjects are ably taught, and on the clinical side, with increase of hospital accommodation and population as great a field of bedside study will exist as at home. We shall indeed fall short of our duty if we fail to turn out as good medical practitioners as graduates in older countries. A medical man should not however be "merely a fellow who prescribes drugs", he should not only have a wide general knowledge of his profession, but he should "make all knowledge his province" and not forget a little dalliance with literature, and when I say he should be a gentleman, I use the word in its fullest, its noblest sense, a sense with which conduct such as the witless larrikinism of last University Commemoration would be utterly incompatible.

Correspondence.

MEDICAL CERTIFICATION.

SIR: A circular from the Medical Board of Victoria concerning medical certification was sent to Victorian doctors recently. It could be construed (judging by the general purport of the circular) that the Chamber of Manufactures is seeking the cooperation of the profession in policing the private activities of patients. The following excerpts could have this interpretation placed upon them:

> In some instances, however, employers have been aware that the patient has not been ill during the period preceding the visit to the doctor. . . .
> A firm sent agents to his surgery. . . .

It is to the credit of the Victorian Branch of the B.M.A. that they emphatically rejected the policy of the Chamber of Manufactures of planting agents upon doctors. The circular does not mention any consultation of the Board with any workers' organizations, e.g., the A.C.T.U., so it would seem that the activities of the Medical Board have been rather one-sided. Correct medical certification is the aim of the profession; but it will not be achieved by the methods adopted by the Chamber of Manufactures or by the threats of the Medical Board of Victoria.

Yours, etc.,

366 Church Street,
Richmond, E.1,
Victoria.
August 26, 1958.

BARRY E. CHRISTOPHERS.

CHLOROQUINE.

SIR: Whilst I am not in a position to comment upon the removal of chloroquine from the free list, Dr. John Poolman's letter (M. J. AUSTRALIA, August 23, 1958) raises the important question of the toxicity of chloroquine.

Chloroquine is usually dispensed in tablets containing 150 milligrammes of base (= 250 milligrammes of the diphosphate or 200 milligrammes of the sulphate). Dr. Poolman's dose of 250 milligrammes per day (misprinted as 520 milligrammes per day) represents 150 milligrammes of base daily or 1050 milligrammes per week. Alving *et alii* (1948)[1] reported visual disturbances, headache, bleaching of the hair, electrocardiographic changes and slight loss of weight in persons taking daily doses of 300 milligrammes of chloroquine (as base) for 77 days. These effects caused no incapacity and diminished or disappeared when administration of the drug ceased. When 500 milligrammes of the base was given weekly for one year occasional headaches and slight loss of weight were observed, and two of thirty persons developed, in the last few months of the test, a lichen planus type of eruption similar to that seen with prolonged administration of "Atebrin" (mepacrine). Chloroquine is used widely for the suppression of malaria, and the dose recommended is 300 milligrammes of base once per week. In the Territory of Papua and New Guinea, however, the amount frequently used is 300 milligrammes of base twice per week, i.e., 600 milligrammes per week. It would be interesting to know the incidence of lichen planus type of eruption in Europeans in Papua and New Guinea and also in cases of rheumatoid arthritis, etc., where these comparatively large amounts of chloroquine may be consumed for long periods.

The visual disturbances associated with increased chloroquine dosage, subjectively described as dizziness, seeing

[1] *J. Clin. Investigation*, 27 (Supplement): 60.

double, blurring of vision on looking from a near to a distant object, or something wrong with the eyes, are obviously important if they concern persons in charge of motor vehicles or aircraft. Acute chloroquine toxicity is seen in a proportion of cases when this drug is used in the treatment of the acute attack of malaria. In adults these effects are usually mild, but when the drug has been given in excessive amounts to infants and young children by injection the results have been fatal. These deaths can be prevented by weighing the child and giving the appropriate amount of chloroquine calculated from the body weight. As an extra precaution the dose could be given in two divided doses with a few hours' interval between them.

Yours, etc.,

ROBERT H. BLACK.

School of Public Health and Tropical Medicine,
University of Sydney,
August 28, 1958.

Post-Graduate Work.

THE MELBOURNE MEDICAL POST-GRADUATE COMMITTEE.

PROGRAMME FOR OCTOBER, 1958.

Visit of Professor Charles Illingworth.

PROFESSOR CHARLES ILLINGWORTH, of the Department of Surgery, Glasgow, will visit Melbourne from October 6 to 13, and will carry out the following programme.

Lectures.

October 7, "Diagnosis and Management of the Jaundiced Patient", at 8.15 p.m., in the B.M.A. Hall, 426 Albert Street; October 10, "Recent Developments in the Treatment of Cancer of the Breast", at 1 p.m., at the Residents' Club, Royal Melbourne Hospital.

Hospital Visits.

October 6, St. Vincent's Hospital, 2 p.m.; October 7, Royal Melbourne Hospital, 9.30 a.m.; October 9, Alfred Hospital, Professorial Unit, 2 p.m.; October 10, Royal Melbourne Hospital: 9.15 a.m., Clinical Research Unit; 11 a.m., professorial ward round.

Visit of Dr. Alice Stewart.

Dr. Alice Stewart, Reader in Social Medicine, University of Oxford, who is well known for her work on the relationship of radiography to the development of leukæmia, will visit Melbourne from October 30 to November 4. Her programme will include the following items.

Lectures.

October 30, "Survey of Leukæmia in Childhood", at 8.15 p.m., in the B.M.A. Hall, 426 Albert Street; October 31, "The Spread of Tuberculosis", at 1 p.m., at the Residents' Club, Royal Melbourne Hospital.

Hospital Visits.

Visits will be made to the Queen Victoria and Royal Children's Hospitals.

Country Courses.

Horsham.

On Saturday, October 4, the following lectures will be given at the Wimmera Base Hospital: 2.30 p.m., "Coronary Disease", Professor R. Lovell; 4 p.m., "Modern Trends in Fracture Treatment", Mr. Eric Price; 5 p.m., Professor Lovell will talk on the activities in his department of medicine; 8.15 p.m., "Recent Advances in Pædiatrics", Dr. Howard Williams.

The local secretary for this course is Dr. R. Webster, Lister House, Horsham. Fees for non-subscribers are at the rate of 15s. per lecture.

Traralgon.

On Saturday, October 11, the following lectures will be given at the Traralgon Hospital: 2.30 p.m., "Surgery of Chest Injuries", Mr. John Hayward; 4 p.m., "Anæsthetic Emergencies and Anæsthesia in Chest Injuries", Mr. T. T. Currie; 8 p.m., quiz session (panel, Dr. Maurice Clarke, Mr. Hayward and Mr. Currie).

The local secretary for this course is Dr. J. E. Joseph, 237 Princes Highway, Morwell. The fee for the course for non-subscribers is £2 2s.

Flinders Naval Depot.

On Wednesday, October 8, at 2.30 p.m., Mr. K. Newman Morris will speak on "Treatment of Thoracic Injuries". This meeting is being held by arragement with the Royal Australian Navy.

Lecture in the Scientific Basis of Medicine.

Dr. J. M. Gardiner will deliver a lecture on "Physiological Aspects of Ventricular Defects" at 8 p.m. on October 3 in the Main Lecture Theatre, Royal Melbourne Hospital. All members of the medical profession are invited without fee.

Information.

The address of the Melbourne Medical Post-Graduate Committee is 394 Albert Street, East Melbourne. Telephone: FB 2547.

Naval, Military and Air Force.

APPOINTMENTS.

THE following appointments, changes, etc., are published in the *Commonwealth of Australia Gazette*, No. 41, of July 24, 1958.

AUSTRALIAN MILITARY FORCES.

Citizen Military Forces.

Eastern Command.

Royal Australian Army Medical Corps (Medical).—2/127045 Captain H. M. Learoyd is transferred to the Reserve of Officers (Royal Australian Army Medical Corps (Medical)) (Eastern Command), 1st June, 1958.

Western Command.

Royal Australian Army Medical Corps (Medical).—To be Captains (provisionally), 29th May, 1958: 5/38095 Sasson Stephen Gubbay, 5/26582 David Norman Spence and 5/26583 Peter Walter Burvill. To be Captain (provisionally), 5th June, 1958: F5/1206 Jocelyn Phillis Mandelstam. To be Temporary Major, 4th June, 1958: 5/26514 Captain R. Paton.

ROYAL AUSTRALIAN AIR FORCE.

Permanent Air Force.

Medical Branch.

Glen Alwyn Tomkins (018879) is appointed to a short-service commission, on probation for a period of twelve months, 18th March, 1958, with the rank of Flight Lieutenant.

The probationary appointment of the following Flight Lieutenants is confirmed: G. C. Nelson (018487), R. J. L. Tebbutt (0211575).

The following Squadron Leaders are appointed to a permanent commission, 1st March, 1958: M. N. McLaughlin (024303), A. Cameron (039928), W. Blake (014736).

Pilot Officer (Student) L. G. Trappett (015935) is granted special leave without pay, 13th March, 1958, to 12th March, 1959, inclusive.

Active Citizen Air Force.

Medical Branch.

No. 25 (City of Perth) Squadron.—Anthony Graham Fisher (051393) is appointed to a commission, on probation for a period of twelve months, 29th March, 1958, with the rank of Flight Lieutenant.

Air Force Reserve.

Medical Branch.

The following are appointed to a commission with the rank of Flight Lieutenant: Geoffrey Richard Trembath Serpell (257979), 27th May, 1957; Werner Stern (257980), 22nd August, 1957.

The provisional appointment of Pilot Officer J. A. Harley (034800) is confirmed and he is promoted to the rank of Flight Lieutenant, 18th December, 1956.

Flight Lieutenant L. N. Walsh (039386) is granted the acting rank of Squadron Leader, 28th April, 1958.

Australasian Medical Publishing Company Limited.

ANNUAL MEETING.

THE adjourned annual meeting of the Australasian Medical Publishing Company Limited was held at The Printing House, Seamer Street, Glebe, N.S.W., on September 17, 1958. DR. W. L. CALOV, the Vice-Chairman, in the chair.

Directors' Report.

The report of the Directors of the Company was as follows:

The Directors submit their report for the twelve months ended June 30, 1958, together with the balance sheet as at June 30, 1958, and the profit and loss account for the twelve months ended June 30, 1958.

It is with deepest regret that we report the death at Sydney on September 6, 1957, of Dr. Mervyn Archdall, Consultant Editor of THE MEDICAL JOURNAL OF AUSTRALIA. Dr. Archdall had retired from the editorial chair on August 31, 1957, after having served on the staff of the Journal for thirty-five years. He had been Editor for twenty-seven years. A plaque will be erected to his memory at The Printing House.

Dr. Ronald Winton has been appointed Editor of THE MEDICAL JOURNAL OF AUSTRALIA, Dr. Arthur M. Gwynn Assistant Editor, and Miss Dorothy Tremlett Lay Assistant Editor. During the year contributions to the Journal covered a wide range of subjects, and continue to be of a high standard.

A satisfactory result was obtained from the year's production of the printing and publishing department, and arrangements have been made for the payment of debenture interest for the year ended June 30, 1958.

New and modern machinery was installed at The Prtining House during the year under review, and is operating satisfactorily.

The company's reserves are used in the business, and we consider the state of the company's affairs is satisfactory.

Dr. W. L. Calov and Dr. J. P. Major retire from office by rotation in accordance with the Articles of Association (Article 39). They are eligible and present themselves for reelection.

H. S. NEWLAND,
Chairman.

September 3, 1958.

Election of Directors.

Dr. W. L. Calov and Dr. J. P. Major were reelected to the Board of Directors.

Notice.

CHILDREN'S MEDICAL RESEARCH FOUNDATION.

THE following is a list of donations to the Children's Medical Research Foundation of N.S.W. received from members of the medical profession during the period from September 3 to 9, 1958.

Dr. and Mrs. J. S. Crakanthorp: £100.
Dr. and Mrs. Henry Sharp: £52 10s.
Dr. Peter Bishop: £50.
Australasian College of General Practitioners (Victorian Faculty), Dr. and Mrs. E. J. Gazzard: £25.
Dr. G. A. M. Heydon: £15.
Dr. and Mrs. N. E. McLaren: £11 11s.
Dr. F. F. Rundle: £11 1s.
Dr. and Mrs. H. J. Emdur, Dr. and Mrs. J. H. B. Christian, Professor H. Priestley, Dr. Z. Wechsler, Drs. Ian and Dorrie Holt, Dr. S. I. Sonabend, Drs. Ross and Elphinstone, Dr. L. T. Milgate, Dr. E. H. Booth, Dr. P. Berger, Dr. and Mrs. W. I. T. Hotten, Dr. and Mrs. James Purchas, Dr. B. E. Frecker, Dr. A. A. McIntosh, Dr. Eric Miles, Dr. V. Hercus, Dr. Marjory Thomas, Dr. J. M. Maclean, Dr. N. F. Leake: £10 10s.
Dr. P. D. Hipsley, Dr. M. O. Kent Hughes, Dr. L. Joseph, Dr. E. Blomfield, Dr. D. B. Morell, Drs. A. B. Yuille and M. J. Brennan, Dr. I. M. Friedman, Dr. I. S. Collins, Dr. E. G. Wilkinson: £10.

DISEASES NOTIFIED IN EACH STATE AND TERRITORY OF AUSTRALIA FOR THE WEEK ENDED SEPTEMBER 6, 1958.[1]

Disease.	New South Wales.	Victoria.	Queensland.	South Australia.	Western Australia.	Tasmania.	Northern Territory.	Australian Capital Territory.	Australia.
Acute Rheumatism	5(4)	3(2)	8(3)	..	..	..	1	..	17
Amœbiasis	..	..	1(1)	..	..	..	..	..	1
Ancylostomiasis	..	..	8	..	..	..	6	..	14
Anthrax	..	..	..	..	..	..	..	..	..
Bilharziasis	..	..	..	..	..	..	..	..	..
Brucellosis	..	1	..	..	..	..	..	..	1
Cholera	..	..	..	..	..	..	..	..	..
Chorea (St. Vitus)	..	1	..	..	..	..	1	..	2
Dengue	..	..	..	..	..	..	..	..	..
Diarrhœa (Infantile)	..	11(11)	9(9)	..	..	..	..	..	20
Diphtheria	..	..	..	..	..	..	..	..	..
Dysentery (Bacillary)	..	1(1)	1(1)	..	1(1)	..	..	..	3
Encephalitis	..	2(2)	..	..	..	..	..	..	2
Filariasis	..	..	..	..	..	..	..	..	..
Homologous Serum Jaundice	..	..	..	..	..	..	..	..	..
Hydatid	..	1(1)	..	..	..	..	..	..	1
Infective Hepatitis	90(37)	20(18)	8(2)	4(1)	..	..	1	..	123
Lead Poisoning	..	..	..	..	..	..	..	..	..
Leprosy	..	..	..	..	..	..	..	..	..
Leptospirosis	..	..	..	..	..	..	..	..	..
Malaria	..	..	..	..	..	..	..	..	..
Meningococcal Infection	..	3(2)	1	..	..	..	..	..	4
Ophthalmia	..	..	..	..	..	..	..	..	..
Ornithosis	..	..	..	..	..	..	..	..	..
Paratyphoid	..	..	..	1(1)	..	..	..	..	1
Plague	..	..	..	..	..	..	..	..	..
Poliomyelitis	..	6(6)	..	..	..	..	..	..	6
Puerperal Fever	..	..	1	..	..	..	..	..	1
Rubella	..	53(42)	..	2(1)	106(97)	1	..	9	171
Salmonella Infection	..	..	..	4(3)	..	..	..	..	4
Scarlet Fever	11(8)	26(18)	3	4(2)	2(1)	..	..	..	46
Smallpox	..	..	..	..	..	..	..	..	..
Tetanus	..	..	2(2)	..	..	..	..	..	2
Trachoma	..	..	..	..	..	..	..	..	..
Trichinosis	..	..	..	..	..	..	..	..	..
Tuberculosis	17(10)	14(13)	5(3)	4(4)	3(3)	1	..	..	44
Typhoid Fever	3(1)	1(1)	..	..	1(1)	..	..	..	5
Typhus (Flea-, Mite- and Tick-borne)	..	..	..	..	..	..	..	..	..
Typhus (Louse-borne)	..	..	..	..	..	..	..	..	..
Yellow Fever	..	..	..	..	..	..	..	..	..

[1] Figures in parentheses are those for the metropolitan area.

Dr. E. R. Marshman: £7 7s.
Dr. and Mrs. Noel Pryde: £5 17s.
Dr. and Mrs. G. J. W. Stump, Dr. D. S. Thomson, Dr. H. G. Royle: £5 5s. 6d.
Dr. N. J. Flynn, Dr. E. A. Hearne, Dr. G. Petit, Dr. P. Watters, Dr. A. J. Lundie, Dr. A. B. Cunningham, Dr. Anthony C. Hathaway, Dr. Colin E. Vickery, Dr. S. John Iceton, Dr. G. E. Hobson, Dr. B. Basil-Jones, Dr. R. C. Chambers, Dr. Eric Campbell, Dr. R. G. Chambers, Dr. Lyle Brown, Drs. I. and E. McKinnon, Dr. Colin Anderson, Dr. K. J. Lazarus, Dr. and Mrs. R. A. Durie, Drs. H. R. Player and N. N. Tereshchenko, Dr. J. W. Lance, Dr. and Mrs. Ashleigh Davy, Dr. F. Grunseit: £5 5s.
Dr. F. McKay, Dr. and Mrs. John Farrell, Dr. John Banfield, Dr. A. H. Hodge, Dr. Ian J. Bryan, Dr. G. E. Jordan, Dr. L. V. Merchant, Dr. G. Archbold, Dr. K. S. Cronin: £5.
Dr. and Mrs. A. M. Hertzberg: £4 4s.
Dr. and Mrs. P. A. Hanks, Dr. M. M. Cranna: £3 3s. 6d.
Dr. Leila Harris, Dr. B. A. Curtin, Dr. J. B. Binks, Dr. Sydney George: £3 3s.
Dr. and Mrs. T. S. Douglas, Dr. J. S. Storey: £3.
Dr. A. S. Waterhouse, Dr. John Church, Dr. and Mrs. R. H. Leach, Dr. and Mrs. D. G. Lampard, Dr. Mary S. Campbell, estate of late Dr. C. H. Northcott, Dr. I. D. Thomas, Dr. T. Oddie, Dr. C. D. Tracey, Dr. I. Monk: £2 2s.
Dr. M. Roland, Dr. D. Van der Poorten: £2.
Dr. T. S. Reeve: £1 11s.
Dr. F. Goldschlay, Dr. Ross and Janice Davis, Dr. G. D. Davis, Dr. and Mrs. J. A. Carnahan, Dr. J. Kalokerinos: £1 1s.
Dr. George Berger: £1.

Total: £836 6s. 6d.

Medical Practice.

NATIONAL HEALTH ACT.

THE following notices are published in the *Commonwealth of Australia Gazette*, No. 50, of September 4, 1958.

NATIONAL HEALTH ACT, 1953-1957.

Notice in Pursuance of Section 134A.

Notice is hereby given that the Medical Services Committee of Inquiry for the State of Queensland, after investigation, having reported on the eighteenth day of June, 1958, concerning the conduct of Samuel Roscoe Stevens, of 553 Brunswick Street, New Farm, a medical practitioner, in relation to his provision of medical services under Part IV of the National Health Act, 1953-1957, I, Donald Alastair Cameron, Minister of State for Health, did on the thirteenth day of August, 1958, reprimand the said Samuel Roscoe Stevens.

Dated this thirteenth day of August, 1958.

DONALD A. CAMERON,
Minister of State for Health.

Notice is hereby given that the Medical Services Committee of Inquiry for the State of Queensland, after investigation, having reported on the eighteenth day of June, 1958, concerning the conduct of Arthur George Harrold, of 553 Brunswick Street, New Farm, a medical practitioner, in relation to his provision of medical services under Part IV of the National Health Act, 1953-1957, I, Donald Alastair Cameron, Minister of State for Health, did on the thirteenth day of August, 1958, reprimand the said Arthur George Harrold.

Dated this thirteenth day of August, 1958.

DONALD A. CAMERON,
Minister of State for Health.

Nominations and Elections.

THE undermentioned has applied for election as a member of the New South Wales Branch of the British Medical Association:

Allison, George Harris Cory, M.B., B.S., 1956 (Univ. Sydney), 16 Hill Street, Harbord.

Deaths.

THE following deaths have been announced:

CLARKE.—Frederick George Joseph Clarke, on September 12, 1958, at Balgowlah, New South Wales.

BLACK.—Michael James Morrison Black, on September 13, 1958, at Singleton, New South Wales.

Diary for the Month.

OCT. 1.—Victorian Branch, B.M.A.: Stawell Oration.
OCT. 1.—Western Australian Branch, B.M.A.: Branch Council.
OCT. 2.—South Australian Branch, B.M.A.: Council Meeting.
OCT. 3.—Queensland Branch, B.M.A.: General Meeting—Jackson Lecture.
OCT. 7.—New South Wales Branch, B.M.A.: Council Quarterly.
OCT. 9.—New South Wales Branch, B.M.A.: Public Relations Committee.
OCT. 10.—Queensland Branch, B.M.A.: Council Meeting.
OCT. 10.—Tasmanian Branch, B.M.A.: Branch Council.
OCT. 13.—Victorian Branch, B.M.A.: Finance, House and Library Subcommittee.
OCT. 14.—New South Wales Branch, B.M.A.: Executive and Finance Committee; Organization and Science Committee.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Medical Secretary, 135 Macquarie Street, Sydney): All contract practice appointments in New South Wales. Anti-Tuberculosis Association of New South Wales. The Maitland Hospital.

South Australian Branch (Honorary Secretary, 80 Brougham Place, North Adelaide): All contract practice appointments in South Australia.

Editorial Notices.

ALL articles submitted for publication in this Journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given: surname of author, initials of author, year, full title of article, name of journal, volume, number of first page of the article. The abbreviations used for the titles of journals are those adopted by the Quarterly Cumulative Index Medicus. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors submitting illustrations are asked, if possible, to provide the originals (not photographic copies) of line drawings, graphs and diagrams, and prints from the original negatives of photomicrographs. Authors who are not accustomed to preparing drawings or photographic prints for reproduction are invited to seek the advice of the Editor.

Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary is stated.

All communications should be addressed to the Editor, THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales. (Telephones: MW 2651-2-3.)

Members and subscribers are requested to notify the Manager, THE MEDICAL JOURNAL OF AUSTRALIA, Seamer Street, Glebe, New South Wales, without delay, of any irregularity in the delivery of this Journal. The management cannot accept any responsibility or recognize any claim arising out of non-receipt of journals unless such notification is received within one month.

SUBSCRIPTION RATES.—Medical students and others not receiving THE MEDICAL JOURNAL OF AUSTRALIA in virtue of membership of the Branches of the British Medical Association in Australia can become subscribers to the Journal by applying to the Manager or through the usual agents and booksellers. Subscriptions can commence at the beginning of any quarter and are renewable on December 31. The rate is £5 *per annum* within Australia and the British Commonwealth of Nations, and £6 *per annum* within America and foreign countries, payable in advance.